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CONTROL AND ERADICATION OF AUJESZKY'S DISEASE: AN ASSESSMENT OF THE SCHEME IN NORTHERN IRELAND.

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LIST OF ABBREVIATIONS

AD	Aujeszky's disease
ADF	Aujeszky's disease free
ADV	Aujeszky's disease virus
CNS	Central Nervous System
dpi	Days post-infection
dpv	Days post-vaccination
DVO	District Veterinary Officer
ELISA	Enzyme-linked Immuno-Sorbent Assay
FF	Farrow to finish
g, gp	Glycoprotein
HSV	Herpes simplex virus
Ig	Immunoglobulin
IN	Intra-nasally
IT	Intra-tracheally
LA	Latex agglutination
LAK	Lymphokine activated killer (cell)
MAb	Monoclonal antibody
MHC	Major histocompatibility complex
MLV	Modified live vaccine
OADF	Officially Aujeszky's disease free
PCFIA	Particle concentration fluorescence immunoassay
PCR	Polymerase chain reaction
PRSS	Porcine respiratory and reproductive syndromme
PS	Primary status
SN	Seroneutralization
TGE	Transmissible gastroenteritis
TK	Thymidine kinase
VN	Virus neutralization (test)

ABSTRACT

Aujeszky's disease (AD) is an important disease affecting the swine industry in many countries. An eradication campaign was officially applied in September of 1994 in Northern Ireland. The current eradication scheme was investigated.

Two main groups of herds were identified from an existing computed database and were classified as: group A, herds that changed their health status, and group B herds that did not change their health status. Herds within each group (A and B) were also classified according to their health status into two groups: a) ***Others,*** herds that were neither infected nor vaccinated and regarded as free of the disease, and b) ***PS's,*** herds that were either vaccinated and/or infected.

The eradication scheme in Northern Ireland is making progress. The linear regressions on months were significantly for the number of herds ($F=29.14$, $P<0.05$), and the high health status OADF ($F=91.5$, $P<0.01$) and ADF($F=167.8$, $P<0.001$). More herds had higher health status in than before. Infected herds decreased from 25 % to 10.7 % after two years. In addition the pig industry expanded with new herds being registered, although their disease status has yet to be assessed.

The groups of herds were analysed by region, current status, herd type and pig population. The main significant difference in the number of herds was found for the comparisons between the groups 1) ***Others*** and ***PS's*** ($\chi^2=116.9$, $P<0.001$), 2) PS1+PS2 and PS3+PS4 ($\chi^2=3.73$, $P<0.05$), and 3) PS1+PS3 and PS2+PS4 ($\chi^2=5.68$, $P<0.01$) of **group A** and **group B** when compared together. It was identified that the most important regions in terms of number of infected herds, pig population and new herds awaiting classification of their health status were UK3 and UK4 where more than 50% of infected herds were found in these two regions that share borders.

1. INTRODUCTION

Aujeszky's disease (AD) or pseudorabies is a contagious disease of pigs which affects the respiratory tract, as well as the central nervous and reproductive systems. AD is caused by a porcine herpesvirus 1, family Herpesviridae, subfamily Alphaherpesvirinae. The economic losses are due to high mortality in new-born piglets, reproductive failure and respiratory problems in growing pigs which delay the time for slaughter.

The disease has been related to areas with high density of pigs, and can be attributed to the intensification of production over the last 20 years. AD can affect swine herds for a long time because after infection the virus tends to become latent and after reactivation, the virus can be shed and transmitted to susceptible animals. Pigs are the major host for Aujeszky's disease virus (ADV) and are the source of infection to other animal species.

Control and eradication programmes against AD are currently active in many countries using different strategies such as depopulation-repopulation, test and removal, offspring segregation, and most important vaccination with gene-deleted-vaccines that, together with the complementary serological tests, allow the differentiation of vaccinated from naturally infected pigs. It is thought that vaccines reduce the clinical presentation of the disease and the amount of virus excretion, thus eventually reducing the virus circulation on pig farms.

Control programmes of AD in many countries have the goal of achieving eradication not only because of herd economic losses but also to enable with international commercial trade in the European Economic area.

Northern Ireland is one of the countries where an intensive vaccination programme with deleted-vaccines together with regular serologic monitoring has been officially applied since September of 1994.

The objectives of this study are:

-To review the geographic distribution of AD in Northern Ireland according to DVO region and pig density areas.

-To review the advances of the AD scheme in Northern Ireland after three years of compulsory vaccination programme.

-To find differences in the number of herds between the group of herds that changed their health status and herds that did not change their health status.

2. LITERATURE REVIEW

2.1. The biology of Aujeszky's disease virus

2.1.1. Description of the virus

2.1.1.1. Morphology

The virus is 150-180 nm in diameter, consisting of a DNA of 75 nm diameter, a nucleocapsid of 105-110 nm with 162 capsomeres. It is an enveloped virus with glycoproteins which are responsible for its immunogenicity (Taylor, 1995).

2.1.1.2. Pathogenicity

The first sites of replication of the virus are the nasal cavity, tonsils and pharyngeal mucosa (Vannier, 1987). The virus is present in the oral and nasal secretions after infection. After replication in the upper respiratory tract it is found in the central nervous system where it causes a non-suppurative meningitis (Maes, Kanitz, and Gustafson, 1983). Cowdry type A intranuclear inclusion bodies are also produced (Baskerville, Mcferran, and Dow, 1973). The excretion of the virus may last 10 to 14 days. The antibody response is seen in serum between 6 to 10 days after infection (Van Oirschot, 1994).

Porcine herpesvirus can replicate in pigs even if they have specific cell-bound antibodies against the virus. However in order to do so, the dose of virus has to be higher than that needed to infect a fully susceptible animal (Smith, 1990).

Alveolar macrophages infected with ADV diminish their effective activity. Other factors that contribute to the suppression of the immune system are the lysis of lymphocytes, focal necrosis in lymph nodes and interference in the synthesis of IL-2 caused by the virus (Iglesias, Pijoan and Molitor,1989; Sakano, Shibata, Samegai and

others, 1993; Hall, Weigel, Siegel and *others*, 1991). This accounts for a higher susceptibility to secondary bacterial infections (Elbers, Tielen, Cromwijk and *others*, 1990; Fuentes and Pijoan, 1987; Muneer, Farah, Newman and *others*, 1988; Chinsakchai and Molitor, 1994).

The virus may infect the uterus causing abortion, foetal death and mummification. Affected boars may shed the virus in semen.

The severity of the disease is influenced by the route of infection, strain, amount of virus, immune status and age of affected pigs. Low virulent strains are characterised by causing subclinical infections, whereas high virulence strains in a susceptible population usually results in a dramatic clinical picture of reproductive failure where mortality in young pigs can reach 100 per cent, although mortality in fattening pigs is generally less than 5 percent (Taylor, 1995).

Pigs recovered from AD wild virus infection are immune but may act as carriers since the virus tends to latency, an important feature of ADV (Van Orischot, 1994; Brown, Osorio and rock, 1990) .

2.1.1.3. Transmission

Pigs are the main host for ADV but they may spread the disease to other susceptible species.

Transmission in pigs is by direct pig-to-pig contact when nasal discharges and saliva contain the cell-bound virus, and the main source of infection is from asymptomatic carrier animals (Donaldson, Wardley, Martin and Ferris, 1983). Air borne transmission plays an important role in the spread of the disease (Christensen, Mousing, Mortensen and *others*, 1990; Mortensen, Mousing, Henriksen and *others*, 1992). Shoenbaum, Beran and Murphy (1990) reported that the infectivity of ADV

decreased up to 50 per cent in less than one hour when the virus was exposed to 20°C and 85 per cent RH. Conversely the survival of the virus in aerosols was increased at 4°C and 55 per cent RH showing that environmental conditions might play a role in the in the viability of ADV. Other ways of spread are mechanical transmission by inanimate objects such as syringes, needles, boots and vehicles.

In epidemiological terms, transmission can be quantified by a reproduction ratio (R_o) which is defined as the mean number of hosts that become infected by one infectious individual. A minor outbreak of disease is considered when $R_o<1$, only a few animals becoming infected. If $R_o\geq1$ then the infection is regarded as a major outbreak. The bigger the R_0 the harder the eradication, but if $R_o<1$ in a certain population or area, then eradication may be achieved (Diekmann, Heesterbeek, and Metz, 1990). In other words, a focus of infection with value $R_0=0$ cannot maintain and spread itself. In a comparison of two different diseases, rubella and measles in the US, one having bigger R_0 (measles) than the other (rubella) before vaccination, it was shown that rubella was certainly eradicated after a compulsory vaccination programme whereas the incidence of measles was only considerably reduced. This example showed the importance of the R_0 value in intensive vaccination programmes (Anderson and May, 1983).

A study carried out by the United States Department of Agriculture (USDA) in 1991 reported that in more than 50 per cent of outbreaks of AD the source of infection was not identified (Annelli, 1991).

2.1.2. Immunology of the virus

2.1.2.1. Cell-virus attachment

The mechanisms for attachment and penetration of ADV into the cell involve different proteins such as gI, gII, gIII, gp63 gp50 and gX (Robbins, Watson, Whealy and *others*, 1986a). The genes encoding for their synthesis have been identified. The biological function of such proteins is described in table 1. The analysis of the genetic sequence has demonstrated that the glycoproteins of ADV are homologues of the ones that are found on the surface of the Herpes simplex virus (HSV), e.g. glycoprotein gII of ADV is the homologue of gB of HSV and gIII of ADV is the homologue of gC of HSV (Robbins, Whealy, Watson and *others*, 1986b; Wathen *et al*, 1986; cited by Ben-Porat, Demarchi, Lomniczi and *others*, 1986) (Table 2). The gIII is one of the non-essential proteins for virus growth *in vitro*, but its role is important in the adsorption of the virus to the cell (Karger and Mettenleiter, 1993). It has been suggested that gIII participates directly in such phenomena or at least it interacts with other viral proteins of the protein complex such as gII and gp50 triggering the adsorption penetration into the cell (Zuckerman, Laszlo and Reilly, 1989).

One of the most important ADV proteins for immunogenicity is gIII, since the avidity of serum from convalescent pigs to target cells that have been infected with deleted strain (gIII-) is less than hyperimmune serum directed to gIII. On accordance with this the neutralising activity of antibodies against ADV seems to be directed to gIII. This point has to be taken into account in the production of gene-deleted vaccines (Zuckermann, Laszlo and Metteinliter, 1990; Ben-Porat *et al*, 1986).

Considering that the cellular mediated immune response by lymphocytes T cytotoxic is directed to gIII, cells expressing gIII as antigen are the main target for effector cells, whereas other glycoproteins like gI, gpIII and gX antigen-expressing cells are less important for cytoxicity. In order to clarify the role of glycoproteins in

the immunogenicity, a study was carried out by Pensaert, Gielkens, Lomniczi and *others* (1992) using monoclonal antibodies (MAbs) showed that MAbs directed to gII, gIII and gp50 neutralised ADV with or without complement, whereas MAbs directed against gI required complement to neutralise the virus. On the other hand glycoprotein gX did not induce neutralising antibodies. Table 3 shows the role of glycoproteins in the immunogenicity for B and T cells, the induction of protection and the action of MAbs.

2.1.2.2. Cell-mediated immune response

The main effector cells of the immune response against ADV are cytotoxic lymphocytes. These cells are responsible for killing viral-infected cells since they recognise viral antigen proteins that are expressed together with major histocompatibility complex (MHC) class I and class II. The former seems to be related to lymphoproliferation through CD_8^+ as a response to live virus (Kimman, Bianchi, De Bruin and *others*, 1995a). Some studies have indicated that the expression of MHC class I can be regulated by the virus (Mellencamp, O'Brien and Stevenson, 1991). However other studies have shown that neither MHC class I nor II are regulated by ADV infection at all (Kimman, Bianchi, De Bruin and *others*, 1995c).

Detection of interferon is possible about 48hrs post infection (Kristien and Pensaert, 1995). The importance of gamma-interferon is related to the activation of lymphocytes. Once infected cells have expressed the viral antigen proteins, they become susceptible for lysis by activated lymphocytes. If lymphocytes are not activated because gamma-interferon is not present, then their killing activity is reduced to a minimal level (Nauwynck and Pensaert, 1995).

The proliferation of T lymphocytes is followed by the production of lymphokines such as interleukin 2. These are responsible for the induction of lymphokine activated-killer cells (LAK) which are responsible for killing ADV-infected target cells (Vanderpooten and Goddeeris, 1995, Kimman *et al*, 1995c).

The identification of the cells that are involved in the lymphoproliferation against ADV was shown by using monoclonal antibodies (MAbs) directed to CD_2^+, CD_4^+ and CD_8^+ . The most important cells were CD_8^+ followed by CD_4^+ (Kimman, De Bruin, Voermas and *others*, 1995b).

Pigs experimentally inoculated with live virus showed lymphoproliferation seven days after inoculation. After a second challenge, the lymphoproliferation could be detected as early as two days, indicating the consequence of induction of memory T cells as an anamnestic proliferation (Kimman, 1992). Unfortunately immunity against herpesvirus is not life-long and after a period of time the virus may replicate again from latent infections (Kimman, De Wind, De Bruin and *others*, 1994).

2.1.2.3. Antibody-mediated immune response

The humoral immune response against ADV is featured by the production of antibodies against virus glycoproteins (Mellencamp *et al*, 1991). IgM appears in serum and secretions at about 6 to 8 days post vaccination (dpv), followed by IgG1 between 10 and 13dpv and IgG2 at about 13 to 20 dpv. The production of IgA in serum, however, tends to be more irregular although not in secretions, since it is the second most important immunoglobulin after IgM, appearing around 10 to 13 dpv in a primary immune response. The duration of high levels of IgG1 and IgG2 in serum may last up to three months, whereas in secretions IgA may be detected for as long as two months in a secondary immune response (Kimman, 1992). In the secondary

response the production of IgG1 and IgG2 in serum is quicker, as early as 3 to 6 days post infection (dpi) and stronger, but nevertheless, IgA is the most important immunoglobulin in secretions (Van Zaane, Browers, Van Oirschot and *others*, 1989; Martin, Wardley and Donaldson, 1986). The role of local immunity (IgA secretory) was demonstrated in a study where surviving pigs from first oro-nasal (ON) experimental challenge did not show a significant cytotoxic activity in peripheral blood cells, suggesting that at the second ON challenge the local immune response was strong enough to neutralise the virus *in situ* (Vanderpooten *et al*, 1995).

Sometimes pigs do not show a secondary antibody response because T cell proliferation suppresses the B cells response by activation of cytotoxic cells, gamma-interferon and tumour necrosis factor (TNF). These factors contribute to eliminate infected cells very quickly and efficiently resulting in poor stimulation of B cells (Kimmann, De Bruin, Voermas, 1996). This explains the fact that protection of pigs against virulent virus after being pre-exposed to the virus may be seen despite low levels of antibodies, indicating the important role of cell immunity (Wittmann *et al*, 1976 cited by Vannier, Hutet and Cariolet, 1995; Pensaert *et al*, 1992).

2.1.2.4. The role of maternal antibodies

The presence of maternally derived immunity must be taken into account in the designing of vaccination schedules, since it has been suggested that passive antibodies interfere with the development of active immune response to vaccine virus (Vannier *et al*, 1995). However, the importance of colostral immunity for protection was demonstrated when pigs having high levels of maternal antibodies survived virulent challenge and had just a mild course of clinical disease (Kimman, 1993).

Kimman (1992) observed the interference of maternal antibodies in the immune response when pigs inoculated intra-nasally (IN) with virulent strain only showed production of IgM in serum and no response was observed in pigs inoculated with avirulent or inactivated vaccine strains. A secondary antibody response was produced only in pigs inoculated with vaccine strains, although higher titres of IgA were developed from pigs IN inoculated suggesting that despite the presence of maternal antibodies pigs can develop an immune response to virulent but not to avirulent or attenuated virus. Maternal antibodies partially suppress an active immune response by negative feed-back of existing IgG (Kimman, 1993), unless a booster dose of vaccine is given (Van Oirschot, Moorman, Berns and *others*, 1991). It was found that the best results occurred when a booster was applied at 16 weeks of age giving it a better chance to overcome maternal antibody interference (Weigel, Lehman, Herr and *others*, 1995).

Studies showing the difference between immune responses of pigs with or without circulating maternal antibodies were carried out by (Vannier *et al*, 1995; Kimman, 1993). Maternal antibodies tend to disappear from systemic circulation at about 10 to 12 weeks of age (Van Oirschot and Gielkens, 1987; Kit,1990).

2.1.3. Latency

ADV is a herpes virus and one of the characteristics of herpes viruses is their capacity to cause latent infections. Many studies have been carried out to better understand this particularity of herpes viruses which accounts for its persistence the pig populations, since reactivation can occur in herds despite vaccination.

Pigs of any age that survived infection may become latently infected, acting as carriers that apparently look healthy. The amount of excreted virus from pigs that have experienced reactivation is enough to infect susceptible animals (Mengeling, Larger, Volz and *others*, 1992).

The main tissues associated with latent infection are the trigeminal ganglia and the tonsils (White, Cicci-Zanella Galeota and *others*, 1996; Brown *et al*, 1990 cited by Chinsakchai *et al*, 1994). The virus was readily detectable by the polymerase chain reaction (PCR) technique for as long as 27 months after experimental infection. Detection of latent infection in different tissues using PCR is shown in table 4. Reactivation of latent virus was achieved by Cowen, Li, Guy and *others* (1990) after the application of dexamethasone (2mg/kg body weight) to animals belonging to herds with history of AD for five years which had been vaccinated with either inactivated or modified live virus (MLV) vaccines. The tonsils were the most successful site for virus recovery.

Piglets of dams with high titre of antibodies are susceptible to wild-type virus and may become carriers despite having high titres of maternal antibodies when treated with dexamethasone (Mengeling, 1989).

It has been suggested that latency-associated transcripts (LATs) are expressed in the CNS (Priola *et al*, 1990 cited by Chinsakchai *et al*, 1994), and the virus may be found after reactivation in non-nerve tissues such as tonsils which are a common site for virus replication.

2.1.4. Virus excretion

Virus circulation is more likely to be found in areas where the pig population density is higher despite the fact that virus excretion is intermittent. The excretion of

virus may be influenced by strain and route of infection since the efficiency of the immune response either locally or systemically will limit this event. In the field, the length of time virus excretion may last longer since infection does not occur at the same time in all animals and the immune status of the herd may limit such phenomena.

Under experimental conditions the virus excretion lasted for 10 to 14 days (Van Oirschot, 1994).

Virulence is a determinant fact: a study reported that pigs inoculated with a virulent NIA-3 strain shed more virus than those inoculated with a mildly virulent Sterksel strain (Kimman, 1993). Conversely when these strains were inoculated into pigs vaccinated with the 783 vaccine strain, the virus excretion of pigs challenged with the milder Sterksel strain was higher than that from those challenged with NIA-3. A possible explanation of these results was the known relationship between 783 strain and NIA-3, the former was stronger because its derived from the latter (Bouma, 1996).

Molecular techniques have been applied for the purpose of diagnosis. For example, to detect latent infections caused by ADV, Mengeling *et al* (1992) suggested the application of the PCR technique in eradication programmes of AD for detection of latent infections in tonsilar biopsies.

Unfortunately, vaccines cannot stop virus excretion (Pensaert, De Smet and De Waele, 1990; Vannier *et al*, 1995; Vannier, Hutet, Bourgeil and *others*, 1991; De Jong and Kimman, 1994) although it has been reported that the amount of virus excreted from vaccinated pigs can be up to 100 to 1000 fold less than non-vaccinated infected ones (Hall *et al*, 1991).

2.2. Vaccines

Vaccines of AD have played a major role in control and eradication programmes in many countries. Differences in the efficiency between vaccines however, have been reported (Vannier *et al*, 1991; Pensaert *et al*, 1992).

2.2.1. Conventional vaccines

Conventional vaccines may interfere with the diagnosis of the disease because we are not able to differentiate vaccinated from infected pigs. In 1987 the use of deleted-vaccines lacking genes coding for expression of glycoprotein I (gI) instead of conventional vaccines was proposed (Van Oirschot *et al*, 1987), which permitted differentiation.

2.2.2. Killed vaccines

Killed vaccines are safer than MLV vaccines because they do not replicate in the animal, although the immune response to this type of vaccines is not as good as that reached with MLV vaccines (Vannier *et al*, 1995).

2.2.3. Gene-deleted-vaccines

Gene-deleted AD vaccines and the complementary serological test that are able to differentiate infected from vaccinated pigs are unique in veterinary and human medicine. The use of these vaccines coupled with the current knowledge of the biology and epidemiology of ADV make this disease a candidate for eradication.

Some of the glycoproteins of ADV that have been so far identified up to now are not essential for virus replication in cell cultures (Pensaert *et al*, 1992; Van

Oirschot, Gielkens, Moorman and *others*, 1990). For example, it has been possible to develop deleted-vaccines lacking of the genes coding for gI, gIII, TK, gX and gp63. For instance in the Bartha strain the genes encoding gI and gp63 are deleted and the inactivation of these deleted proteins have reduced the virulence for pigs (Van Oirschot, 1994). The 783 strain, derived from the highly immunogenic NIA-3 allows the differentiation between vaccinated and infected pigs (Van Oirschot *et al*, 1991). However, manipulations of these glycoproteins may interfere with the immunogenesis and replication of the virus in vaccinated animals since some glycoproteins are more important than others (Van Oirschot *et al*, 1990). For instance TK is not essential for virus growth in tissue culture, but it is important as a virulence determinant promoting virus replication in non-dividing cells e.g. nerve cells (Pensaert *et al*, 1992).

The success of gene-deleted vaccines has been due to the selection of the most important glycoproteins which are consistent and efficacious markers for differentiation.

All the current deleted-vaccines have been evaluated in pigs, and the results have shown that these types of vaccine are more consistent than conventional vaccine types. We still do not know which deleted-vaccines are best since different factors such as breed, age, dose, period of time between vaccination and challenge, virus strain and deleted protein, route of administration and method of evaluation may influence this performance (Van Oirschot *et al*, 1990; Osorio, 1995). Today there are a wide range of vaccines commercially available with different combination of deleted glycoproteins (Table 5).

2.2.3.1. Recombination

There is a general concern about the likelihood of virus recombination between MLV and wild-type virus in the field, although MLV vaccines have been applied in many countries in intensive vaccination programmes for a long time and no recombination has been reported so far. The risk of recombination is relatively low because genetically engineered gene-deleted vaccines lack virulence genes, although this risk cannot be ignored. Theoretically, recombination seems feasible, but to make possible the exchanging of genetic material both viruses must infect the same cell at the same time which is quite unlikely since cellular receptors may be blocked already by the first infected virus.

Another point that supports the idea of low risk of recombination was argued by Donaldson (1984) who stated that the replication of attenuated vaccine virus was restricted just to the site of application (e.g. neck region) and no vaccine virus was shed in natural secretions after vaccination.

Van Oirschot (1992) suggested that gene-deleted vaccines must manifest absence of:

-vaccine virus transmission

-reactivation of latent vaccine virus

-recombination with other ADV strains

-pathogenicity for non-porcine host

-establishment in the pig population

in order to diminish the risk of recombination.

2.2.4. Ideal vaccine

An ideal vaccine for the control of AD should be able to: induce both humoral- and cell-mediated immune responses; completely reduce virulent virus excretion; allow the proper differentiation of vaccinated from infected pigs, and prevent latent infections (Pensaert *et al*, 1992).

The goal of AD vaccines is to prevent the circulation and perpetuation of wild-type virus. It was known that all vaccines against AD were not able to prevent this. However new studies have demonstrated that deleted-vaccines with higher colonisation avidity (Strain SMB gX-, TK-, and gI+) are able to prevent infection with wild-type virus by pre-colonisation tissues targeted in latent infection e.g. trigeminal ganglia (Osorio, 1995).

2.3. Vaccination

Vaccination as a measure of control of AD has been widely used throughout the world for several years. At the moment many countries such as Northern Ireland, The Netherlands, Germany, France, Mexico, USA and others are using this approach to eradicate the disease.

Vaccination has two main purposes: a) to protect individuals against the clinical presentation of the disease i.e. individual immunity; and b) to reduce the chance of becoming infected when the individual is part of a vaccinated population i.e herd immunity. The achievement of herd immunity after vaccination is more desirable than individual immunity and is a good indicator of the efficacy of vaccination (De Jong *et al*, 1994).

Although vaccination does not entirely prevent infection, it significantly reduces the virus circulation and the clinical presentation of the disease (Vannier *et al*, 1995; Pensaert *et al*, 1992). The importance of mass vaccination is that it reduces

the number of cases. It does not matter if the vaccination does not cover 100 per cent of the targeted population since the susceptible population tends to be smaller as the proportion of vaccinated animals grows (Anderson, 1992).

When monitoring a vaccinated area, particular attention should be paid to major outbreaks, since minor outbreaks are less important for the dissemination of the virus (Van Nes, Stegeman, De Jong and *others*, 1996). The detection of minor outbreaks of AD is however, important for determining whether or not to stop vaccination. Only when the monitoring of the disease has been done regularly and the risk of infection has become very low, then it is recommended to stop vaccination (Stegeman, Elbers, Loefen and *others*, 1996; Vannier, Vedeau and Allemeersch, 1997).

The strategy based on the combination of vaccination and test and removal seems to be one of the best approaches in areas where the disease is endemic rather than a stamping-out policy, which is not economically advisable at present (Pensaert *et al*, 1990).

The efficacy of vaccines has been assessed by the reduction of virus transmission by De Jong *et al* (1994). His results showed reduced virus transmission amongst pigs vaccinated with the 783 strain, compared to unvaccinated ones. However several studies have reported virus titres and duration of virus excretion after challenge in vaccinated pigs (McFerran, McCracken and Dow, 1982; Van Oirschot *et al*, 1987; Pensaert *et al*, 1990).

It seems that the best immunogen is the wild-type virus since pigs recovered from natural infection are resistant to experimental challenge unless a very high dose of virulent virus is applied by ON and intra-tracheal (IT) inoculations (Miry and Pensaert, 1988 cited by Pensaert *et al*, 1990).

In a comparative study in which different types and methods of vaccination were studied, it was demonstrated that the most effective vaccine was the one based on MLV when administered IN. This was assessed by a measurable decrease in virus shedding and a milder clinical picture of the disease which was reflected in an earlier recovery of the affected animals (Mengeling *et al*, 1992). Because the ON cavity is the most important natural route of infection for ADV, local levels of IgA might account for the superior performance of this method of vaccination, since intra-muscular application does not trigger a significant nasal antibody response (Weigel *et al*, 1995).

A comparison between inactivated and MLV vaccines using two applications of the former showed that inactivated vaccines were not effective in reducing virus excretion despite the fact that two inoculations were applied compared to a single shot of MLV vaccines. However in spite of their cheaper price, MLV vaccines have not yet been widely accepted in some countries due to the fear recombination with wild-type virus. The performance oil-in-water suspended and phosphate buffered saline (PBS) vaccines was compared, resulting in lesser amount and excretion of virus with the former. This suggested that the vaccine vehicle plays a role in the immune response (Vannier *et al*, 1991; Pensaert *et al*, 1990).

2.3.1. Vaccination schedules

There are many vaccination schedules reported in the literature using different type of vaccines e.g. MLV or Killed, adjuvant phosphate buffered solution (PBS) or oil-in-water and different deleted proteins. The designing of an ideal vaccination programme should take into account aspects related to the prevalence of the disease in the herd and area where the herd is located, duration of maternal antibodies and

presence of virus circulation. Some programmes are designed to vaccinate just the breeding stock, others include also fattening pigs. The question is whether to apply one or two shots in the fattening ones. To this point Stegeman, Van Nes, De Jong and *others* (1995) reported that the incidence of infection in pigs vaccinated once was higher (38 per cent) than the incidence of pigs vaccinated twice (10 per cent) giving values R_o=3.4 and R_o=1.5 respectively. Vannier *et al* (1991) argued that if vaccinated pigs were younger than 8 weeks of age a booster shot enhanced the immune response.

2.3.2. Vaccination experiences in the control of ADV

It seems that control of AD in large herds (>400 sows) requires the implementation of husbandry measures since simple application of vaccine is not enough to stop virus circulation. Field studies in large herds with high seroprevalence of AD have reported considerable reduction and elimination of infected animals after 1 to 3 years of intensive vaccination programmes combined with changes in management (Lehman, Weigel, Seigel and *others*, 1993; Lehman, Weigel, Seigel and *others*, 1994; Engel and Weirup, 1989). However other studies in endemic areas have shown that even after intensive vaccination for as long as three years, virus circulation was not fully stopped, since seropositive reactors being still detected. Fortunately no major outbreaks were reported in such areas, indicating that vaccination reduced markedly the number of susceptible animals (Stegeman *et al*, 1996; Stegeman, Van Oirschot, Kimman and *others*, 1994; Van Nes, Stegeman, De Jong and *others*, 1997).

The husbandry measures that help in the reduction of virus circulation for the control of AD include depopulation/repopulation, test and removal of seropositive

reactors, offspring segregation with or without off-site rearing of replacement gilts, limited movements of pigs within the herd, reduced number of pigs per pen, and all-in-all-out pig flow practice. Any of these strategies may be applied together with vaccination (Hall *et al*, 1991).

2.4. Diagnostic tests

The diagnosis of a disease in an eradication campaign is crucial.

A wide range of serological tests are available for detection of antibodies to ADV. Some of them are regarded as screening tests which detect antibodies to the whole virus, such as seroneutralisation (SN), enzyme-linked immunosorbent assay (ELISA), latex agglutination (LA), and particle concentration fluorescence immunoassay (PCFIA), whereas others as regarded as differential, based on ELISA which detect antibodies directed to a variety of ADV glycoproteins e.g. gI, gIII or gX.

Serological testing is the most suitable diagnostic procedure for eradication programmes of AD. The application current gene-deleted-vaccines enables the differentiation of the immune response of pigs to either the wild-type or vaccine virus, using the recently developed ELISA (gI) test (Van Oirschot, Rhiza, Moonen and *others*, 1986).

A comparison between ELISA gI, conventional ELISA and two SN tests (SN 1h and SN 24h) showed that the ELISA gI had as high sensitivity (99.2%) and specificity (100%) as the conventional ELISA; the two SN (1 and 24 h) had similar values for these parameters but the ELISA gI could detect a lower titre of antibodies than the conventional ELISA. The earliest test for detecting antibodies were the SN tests (7 dpi) and the latest the ELISA gI (14 dpi) (Van Oirschot, 1991). In 1981 Todd, McNair, McNulty and *others* developed a conventional ELISA. The comparison

between this test and a microtitre SN test indicated that the ELISA was more sensitive and detected antibodies earlier (7 dpi) than the SN. The ELISA has shown some advantages such as being quicker and cheaper. Moreover it could be automated thereby, be suitable for testing large numbers of sera.

The sensitive value for different differential ELISA tests was reported by White *et al* (1996) after 27 months of experimental infection (Table 6).

Recently developed serological tests e.g. differential ELISA have supplanted SN tests which were regarded as the "gold" standard test for AD and demonstrated that they are equal or even more sensitive. Nowadays, differential ELISA tests are available as commercial kits for testing thousands of samples for the monitoring and surveillance of AD which is fundamental in eradication programmes (Toma and Eloit, 1989).

2.5. Epidemiological aspects

2.5.1. Risk factors for the disease

One of the basic principles in epidemiology is that diseases are not randomly distributed in a population since casual factors such as spatial, temporal and demographic characteristics are involved in determing the patterns of disease (Hungerford, 1991).

Epidemiological studies have been focused on trying to identify risk factors that play a role in the presentation and persistence of AD. These factors include environmental conditions e.g. location, prevailing wind patterns and herd characteristics e.g. herd size, density, husbandry measures.

2.5.1.1 Geographical studies

A region with a low herd density has better chances to become free of the disease in a shorter period of time than a region with high herd density (Buijtels, Huirne, Dijkhuizen and *others*, 1997).

Studies have found that herds located within an area with a low density of vaccinated herds are 2.7 times more likely to become infected than those herds located in an area with a higher density of vaccinated herds (Norman, Sischo, Pitcher and *others*, 1996), reflecting the importance of area vaccination in reduction of virus circulation since R_o tends to be lower in vaccinated herds (Smith 1990).

Other studies have found that the proximity of a herd to a river or lake (within 1 km) reduced the risk of infection [relative risk (RR=0.52)] while proximity to herds (within 5 km radius) practising farrow-to-finish and complete confinement (RR=2.12 and 3.42, respectively) increased the risk of infection. These results suggested that more important risk factors were those related to herd type rather than environmental features (Marsh, Damrongwatanapokin, Larntz and *others*, 1991).

2.5.1.2. Characteristics of the herd

There are herd factors already identified that have been related to virus circulation, since these characteristics play an important role in the risk of a herd to become infected with AD. Amongst these factors concerning to management practices are open or closed herds, herd type, and degree of confinement. Also factors such as pig density of certain area and the immune status of pigs may interact one to each other to determine the dynamics of ADV. Indeed, it has been reported that the most important factors for persistence of ADV are herd size and pig density (Smith, 1990).

The prevalence of the disease is regionally distributed since farms with high prevalence of the disease are clustered geographically as are farms with low prevalence. For example, it has been found that spread of AD was due to the proximity to an infected herd with high prevalence (28-30%) of the disease (Austin and Weigel, 1992).

In a field survey carried out by Norman *et al* (1996) to identify risk factors for type of herd it was reported that the risk was 2.8 times more likely of becoming infected for a farrow to finish (FF) herd than the one for a feeder-pig-finish type.

Another study found that the spread of ADV in breeding stock was 6.9 times more when the herd had common housing for gilts and sows together with a herd size of >400 sows and had constant seropositive finishing pigs (Duffy, Morrison and Thawley, 1991).

Vaccinated herds are less likely to become infected but if so the amount of excreted virus tends to be lower in vaccinated herds than in unvaccinated ones. Therefore, a R_0 value less than 1 can be expected, as was demonstrated by (Smith, 1990; Van Nes *et al*, 1996).

Table 1. Identified genes and biological characteristics of ADV glycoproteins.

Gene	*Protein*	*Size (kDa)*	*Essential*	*Function*	*Virulence*
gII	913aa	110-68-55	+	penetration	N.A.
gIII	479aa	92	-	adsorption	+
TK	320aa	35	-	thymidine kinase	+
gH	686aa	84	+	penetration	N.A
PK	336aa	41	-	protein kinase	+
gX	498aa	99	-	unknown	-
gp50	402aa	60	+	penetration	N.A.
gp63	350aa	63	-	unknown	+
gI	577aa	110	-	unknown	+
11K	106aa	11	-	tegument	?[b]
28K	256aa	28	-	unknown	-

[a]N.A= not applicable

[b]= Not data available

Source Pensaert *et al*, 1992.

Table 2. Homologues between ADV and HSV.

ADV	**HSV**
gI	gE
gII	gB
gIII	gC
gp50	gD
gp63	gI
gX	gD
gH	gH
11K	US9

Source Van Oirschot *et al*, 1990.

Table 3. Role of ADV proteins in immunogenecity and protection.

Property	gI	gII	gIII	gX	gp50	gp63
MAbs neutralise[a]	-	+	+	-	+	-
MAbs neutralise+C'	+	+	+	-	+	-
Netralising activity in pig serum[b]	-	-/+	+	-	+	-
MAbs pig protection[c]	-	+	+	-	+	np
Antigen for						
B cells	+	+	+	+	+	?
Tc cells	-	?	+	-	?	-[d]
Induction of protection	±	++[e]	++	-	++[e]	±

[a]MAbs neutalised or did not neutralise ADV with or without complement.
[b]Neutralising activity in pig serum.
[c]MAbs protected or did not protect pigs against challenge.
[d]Protein kinase and gp63 have not been reported as target antigens for antibodies of pigs.
[e]gII, gH ÆE and gp50 are essential for virus replication and thereby for the induction of immunity by live vaccines.

Adapted from Van Oirschot *et al*, 1990, and Pensaert *et al*, 1992.

Table 4. Detection of latenf infection with ADV by PCR in tissues collected by tonsillar biopsy at 12 weeks after vaccination and necropsy at 19 weeks after vaccination.

No. of pigs	Tonsilar biopsy	Trigeminal ganglia	Olfactory bulbs	Tonsils
16	3*	16	11	2
10	0	10	1	1
9	1	9	7	1
10	0	9	1	0
10	0	9	0	0
Total	4	53	20	4

*No. of pigs in which PCR revealed ADV infection
Source Mengeling *et al*, 1992.

Table 5. Deleted proteins in commercial AD vaccines.

	Gene deleted				
Vaccine	**gI**	**gp63**	**gIII**	**gX**	**TK**
Suvaxyn	-	+	+	+	-
Nobi-Porvac	-	+	+	+	-
Tolvid	+	+	+	-	-
Omnimark	+	+	-	+	-
PRV-marker	+	+	+	-	-

Adapted from Wittmann, G. and Rziha, H. 1989.

Table 6. Sensitivity of serologic tests for ADV.

Test	Sensitivity (100)
Screening	
SN (1:2)	100.0
SN (1:4)	96.4
ELISA (conventional)	100.0
LA	100.0
PCFIA	98.1
Differential ELISA	
gI	100.0
gIII	100.0
gX	94.5

Adapted from White *et al*, 1996.

3. MATERIAL AND METHODS

Information was collected from an existing database of the pig health system (PHS) of the AD scheme officially running in Northern Ireland since September of 1994.

3.1. Background information of AD in Northern Ireland.

A serological survey for AD took place in Northern Ireland between 31-01-93 and 19-11-93. The sample included 2339 breeding sows from 187 herds, 6237 fattening pigs, and 2327 culled breeding animals at slaughter. The survey revealed that 25% of farms, 13.5% of fattening pigs, 13.6% of culled breeding animals, and 7.9% breeding animals were infected. The prevalence was higher in areas with higher pig density.

3.2. Classification of herds.

The AD scheme has classified the existing herds into three main categories according to their health status. The status of herd is given by the Department of Agriculture based on all evidence available and in close collaboration with the farmer, private practitioner and DVO. The categories are:

I. OADF-Officially Aujeszky's disease-free is regarded as the highest health status where the following conditions have been met:

1.There are no pigs in the herd vaccinated against the disease.

2.The breeding pigs must have undergone testing for AD, with negative results;

(a) two samplings of 100 % of the breeding pigs by the conventional ELISA(whole virus) between 6 to 8 months apart to achieve this health status; or

(b) samples must be taken from 10% of all breeding pigs kept on the herd subject to a minimum of 6 samples and a maximum of 40 at 6 monthly intervals in the first year after achievement OADF status and thereafter annually. The samples must be tested for the presence of antibodies to the **whole virus** with negative results. The sampling must include samples from at least two stock boars and from sows and gilts of different age groups and, in the case of a herd comprising more than one site, from all the sites where pigs are kept.

(c) after the first two qualifying samplings only pigs from an OADF herd may be moved into the herd.

II. ADF-Aujeszky's disease-free and regarded as a middle health status where the following conditions have been met:

1.All pigs kept on the herd must have been inoculated with deleted-vaccines.

2.The breeding pigs must have undergone testing for AD with negative results;

(a) two samplings of 100% of the breeding pigs by the differential ELISA (**gE**) between 6 to 8 months apart;

(b) samples must be taken from 10% of all breeding pigs kept on the holding subject to a minimum of 6 samples and a maximum of 40 at 6 monthly intervals in the first year after of granting ADF status and thereafter annually. The samples must be tested for antibodies to **gE** with negative results. The sampling must include samples from at least two stock boars and from sows and gilts of different age groups and, in the case of a holding comprising more than one site, from all the sites where pigs are kept.

(c) after the first two qualifying samplings only pigs from an ADF or OADF herd may be moved onto the herd

III. PS-Primary status is regarded as the lowest health status and is the initial category when a new herd is registered and included by the first time in the pig health system (PHS). The PS category is divided into subcategories as follows:

PS*: downgraded herd but it may go up again.

PS1: all tested pigs show evidence of vaccination but no evidence of infection.

PS2: all tested pigs show evidence of vaccination and some evidence of infection.

PS3: not all tested pigs show evidence of vaccination but not apparent infection.

PS4: not evidence of vaccination but infected.

PCA: not yet tested for antibodies, may have vaccinated pigs, candidate ADF (c ADF).

PCO: not yet tested for antibodies, no vaccination, candidate OADF (c OADF).

3.3. Movement control.

The permitted movements of pigs vary according to the disease status of the herd and are identified by colour-coded documents as follows: OADF-green, ADF-amber and PS-red.

3.4. Vaccination schedule.

In accordance with the Aujeszky's Disease Scheme Order Northern Ireland

1994, all the vaccines used for the purpose of eradication are gene **gE**-deleted-vaccines (Bartha strain). Herds are required to be vaccinated as follows:

-breeding stock, three times per year at 4 monthly intervals.

-fattening pigs and replacement breeding stock, at 10 and 14 weeks of age.

-breeding stock not previously vaccinated, twice 4 weeks apart and include them in herd schedule thereafter.

-pigs are not required to be vaccinated if they are kept either on OADF or ADF herds or by officially exempted.

3.5. Serological testing.

Two ELISA tests are used for serological testing in Northern Ireland, one is used as a screening test (whole ELISA) developed at the Veterinary Sciences Division, Stormont, Belfast (NI) as described by Todd (1981) which detects antibodies either to wild-type virus or vaccine virus. Depending on the source of samples (OADF, ADF or PS herds) and the results of this first screening test, the samples can be tested by a second test which is differential ELISA (gE) (Herdcheck®, Idexx S.A. Cedex France).

A positive reaction to the whole ELISA means that the sample has antibodies either to the wild-type virus or vaccine virus. If the sample gives a negative reaction it means that the animal has no antibodies; in other words, the animal has not had contact with the virus and is regarded as free of infection. Positive samples shall be tested with the differential ELISA (gE) in order to discriminate between wild-type virus and vaccine virus specific reaction. A positive reaction to the ELISA (gE) means that the sample has antibodies to the wild-type virus and then the pig is regarded as infected.

All blood samples are tested by the Veterinary Services Division Stormont and are free of charge for the farmer. The blood samples are used for other survey e.g. transmissible gastro-enteritis (TGE) and porcine reproductive and respiratory syndrome (PRRS).

3.6. Pig health system (PHS)-Identification of herds.

All pig herds are required to be registered and issued with a licence by the Department of Agriculture. Herds are identified according to the DVO region, the identification denotes the region within which the herd is located. For example, UK1 ABC denotes a herd within division number 1 which is Armagh. Northern Ireland is divided into 10 DVO regions (Appendix 1).

3.7. Database.

The PHS is a computed database for the administration of all herds and markets and is continuously updated with the following information:

-identification herd number

-health status

-number of pigs

-veterinary practitioner

-vaccination

-testing results

The information collected from the database for the analysis in this dissertation was updated up to the 7th of August 1997.

Herds were examined by region, current status, herd type, and pig population.

Herds were divided into two main groups: **group A**: herds that changed their

health status, 667 records, and **group B**: herds that did not change their health status, 840 records.

For the analysis of the data herds within groups were also divided into two groups according to their current health status: a) the group ***Others*** included: ADF, CA, CO, OADF, PCA and PCO herds (Appendix 2 describes current health status). These herds were neither vaccinated nor infected and were regarded as free from the disease since they had already stopped vaccination and were awaiting for a higher health status change. However herds could also be downgraded if seropositive animals were detected (it is important to have in mind that the scheme is a dynamic process). b) the group ***PS's*** included all PS subcategories: PS, PS*, PS1, PS2, PS3 and PS4 (Appendix 2). ***PS's*** herds were the target group to be compared with since they were the ones which are dealing either with the disease or vaccination.

PS's herds were classified as follows:

PS1: vaccinated, not infected.

PS2: vaccinated, infected.

PS3: not vaccinated, not infected.

PS4: not vaccinated, infected.

PS1+PS2: vaccinated

PS3+PS4: not vaccinated

PS1+PS3: not infected

PS2+PS4: infected

Statistical differences between **group A** and **group B**, and group ***Others*** and group ***PS's***, and ***PS's*** (one to each other) were looked at using non-parametric tests (Chi-square and Mann-Whitney) on Epi Info software Version 6.

Chi-square test was used for the comparison between groups: ***Others*** and

PS's, and *PS's* (one to each other) by the variables region, current status, and herd type from **group A** and **group B** respectively, and also for **group A** and **group B** together. The numbers of observations in each group were pooled to avoid values <5 in cells enabling the test to be valid.

The pig population could not be analysed by parametric test because the data showed a non-normal distribution. This variable was analysed by Mann-Whitney test for differences between **group A** and **group B** together by region.

4: RESULTS

4.1. Advances of AD scheme in Northern Ireland

4.1.1. Figures of the pig population and its serologic condition reported from 1994 to 1996.

At the beginning of 1996 the pig population in Northern Ireland contained 700,000 pigs in 3,000 herds. The number of pig farms increased after the "BSE crisis", created a move from cattle farming into a pig farming, although the number of pigs remained almost the same. The number of farms increased from 2,733 by the end of 1994 to 3,707 by the end of 1996 (Table 7 and fig. 1).

The increment in the number of pig farms resulted in a bigger number of primary status (PS) since 1144 herds were classified as PS herds in 1994 compared to 1536 at the end of 1996 giving a difference of 392 more herds. There was also a marked increase in OADF and ADF herds giving differences of 674 and 120 more herds respectively. This is explained by the reduction in the number of cOADF, PS/c OADF, cADF and PS/cADF herds respectively, since the dynamics of changing health status was attributable to the advances of the scheme (Table 7).

The linear regression was significantly different for the total number of herds ($F=29.1$, $P<0.05$), and the categories OADF ($F=91.5$, $P<0.01$) and ADF herds ($F=167.8$, $P<0.001$) according to six monthly intervals. More herds appeared in both categories, although one increased more rapidly than the other (Table 7 and 7a).

The total number of PS herds by the end of 1996 was 1536. The distribution of herds by region showed that the highest percentage of this herd category was found in region UK1 and UK3 followed by UK8. An estimation of 288 infected herds was reported (Table 8 and fig. 2). Infection decreased in almost every region

except in regions UK5 and UK9.

The percentage of herds according to their serologic status showed an increase of OADF herds to 26 % by the end of 1996 compared to 2 % in 1994 which is attributable to a decrease of Candidate herds from 46 % to 13.3 % herds. The percentage of PS herds slightly increased from 51 % in 1994 to 56 % in 1996 as a result of the increase in total number of operational herds from 2242 in 1994 to 2725 by the end of 1996 (Table 9 and fig 3).

By the end of 1996 a total of 1536 herds were regarded as PS (including subcategories) herds which represented 56 % of the total number of operational herds (2725). Samples from 517 (33.6 %) out of 1536 herds of this category were collected and examined, 134 were found infected (PS2 and PS4 together) which represented 8.7 % of the total number of herds of this category. The remaining 1019 (66.4 %) PS herds were not sampled (Table 10, fig 4 and 4a).

The seroprevalence of the disease decreased from 25 % to 10.7 % infected herds after two years since the AD scheme in Northern Ireland officially started.

4.1.2. Analysis of group A and group B herds, and *Others* and *PS's* herds.

4.1.2.1. Analysis by region.

The distribution of herds for **group A** by region showed that region UK8 had highest number of herds (nearly 200) representing 30 % of the total number of herds. Regions UK1 had 154 and UK3 had 105 herds representing 23.1 % and 15.7 % respectively of the total of herds (Table 11, map 1).

The distribution of herds for **group B** showed that the highest number of herds was found in region UK4 with 211 herds representing 25.1 % of the total number of herds. Regions UK3 had 176 and region UK9 had 105 herds representing

21.0 % and 12.5 % respectively of the total number of herds (Table 12, map 1).

4.1.2.2. Analysis of herds by current health status.

The distribution of herds by current status of **group A** showed that 187 (28.0 %) had PS3 status, 133 (19.9 %) had OADF status, and 97 (14.5 %) had PS1 status (Table 13).

The distribution of herds for **group B,** 359 (42.7 %) had PS status, 154 (18.3 %) had PS3, and 109 (13.0 %) had PS1 status (Table 14).

4.1.2.3. Analysis of herds by region and current health status.

The analysis of herds by region and current health status of **group A** from the three regions with the highest number of herds showed that 77.5 % of herds of region UK8, 63.8 % of herds of region UK3, and 62.3 % of herds of region UK1 were ***PS's*** herds (Tables 15 and 15a; map 2).

Significant differences in the number of herds were found by chi-square test in the comparisons between the following groups:

Others and ***PS's*** (χ^2=66.01, P<0.001)

PS1 and *PS2* (χ^2=15.2; P<0.001)

PS1+PS3 and *PS2+ PS4* (χ^2=14.69, P<0.001)

Conversely the comparisons were not significant between the groups:

PS1 and *PS3*

PS3 and *PS4*

PS2 and *PS4*

PS1+PS2 and *PS3+PS4* (Table 15b).

The analysis of herds by region and current health status of **group B** from the

three regions with the highest number of herds showed that 97.1 % of herds of region UK3, 89.5 % of herds of region UK4, and 72.3 % of herds of region UK9 were ***PS's*** herds (Tables 16 and 16a; map 2).

Significant differences in the number of herds were found by chi-square test in the comparisons between the following groups:

Others and ***PS's*** (χ^2=51.91, P<0.001)

PS1 and *PS3* (χ^2=17.88, P<0.001)

PS2 and *PS4* (χ^2=6.74, P<0.03)

PS1+PS2 and *PS3+PS4* (χ^2=37.04, P<0.001)

Conversely the comparisons were not significant between the groups:

PS3 and *PS4*

PS1+PS3 and *PS2+PS4* (Table 16b).

When **group A** and **group B** were pooled and compared together significant differences in the number of herds were found by chi-square test for the comparisons between the groups:

Others and ***PS's*** (χ^2=116.93, P<0.001)

PS1+PS2 and *PS3+PS4* (χ^2=3.73, P<0.05)

PS1+PS3 and *PS2+PS4* (χ^2=5.68, P<0.01) (Table 16c).

Comparisons in the number of herds by chi-square test between the groups ***Others*** and ***PS's*** of **group A** and **group B** inter-regionally showed significant differences (P<0.05) for all the regions except those between the regions [UK0 and UK6], [UK2 and UK5], [UK2 and UK7], [UK2 and UK9], [UK6 and UK8], and [UK7 and UK9] (Table 17).

Comparisons in the number of herds by chi-square tests found significant

differences between the groups ***Others*** and ***PS's*** of **group A** and **group B** intra-regionally as follows:

UK0 (χ^2=11.48, P<0.001)

UK1 (χ^2=6.93, P<0.003)

UK3 (χ^2=62.29, P<0.001)

UK4 (χ^2=7.38, P<0.01)

UK6 (χ^2=5.27, P<0.02)

UK9 (χ^2=5.90, P<0.009) (Table 17).

4.1.2.4. Analysis of herds by herd type.

The analysis of herds by herd type of **group A** showed that only 348 (52.1 %) out of 667 records had recorded the herd type. The most common herd type was birth to weaning (BW) (Appendix 3 gives a description of herd types). There were 227 BW herds (65.2 %) and 205 (90.3 %) of these were found within the group ***PS's***.

Birth to bacon (BB) was the second most common herd type with 78 herds (22.4 %) of which 69 herds (88.4 %) belonged to ***PS's*** herds (Tables 18, 19 and 19a).

The comparison between ***Others*** and ***PS's*** was not significantly different.

The analysis of herds by herd type of **group B** showed that only 270 (32.1 %) out of 840 records had recorded this variable. The most common herd type was also BW represented by 147 herds (54.4 %) that had this herd type. Of these, 141 herds (95.9 %) belonged to the group ***PS's***. Birth to bacon (BB) was the second most common herd type represented by 86 herds (31.9 %) that had this herd type. Of these, 78 herds (90.6 %) belonged to group ***PS's*** (Tables 20, 21 and 21a).

The comparison between the groups ***Others*** and ***PS's*** by herd type was not

significantly different.

4.1.2.5. Analysis of herds by pig population.

The analysis of herds by pig population of **group A** revealed that the regions with the highest number of pigs were UK1 with 37,611, UK8 with 31,744, and UK0 with 22,749 pigs (Table 22).

The analysis of herds by pig population of **group B** revealed that the regions with the highest number of pigs were UK4 with 52,628, UK3 with 27,102, and UK2 with 22,828 pigs (Table 23).

The intra-regional comparison of pig population by Mann-Whitney test between **group A** and **group B** showed significant differences only between regions UK1 ($U=68.5$, $P<0.04$), and UK6 ($U=413$, $P<0.001$). The remaining intra-regional comparisons between **group A** and **group B** were not significant (Table 23a).

The analysis of herds of **group A** showed similar pig population for the groups Others and PS's, being 90,353 and 99,573 pigs respectively. The pig population for the groups PS2, PS4 (infected herds), and PS herds was 12,666, 11,488, and 9,725 pigs respectively (Table 24).

The analysis of herds of **group B** showed a big difference of the pig population for the groups ***Others*** and ***PS's***, being 8,473 and 136,951 pigs respectively. The pig population for the groups PS2, PS4 (infected herds), and PS was 15,954, 20,551, and 32,551 pigs respectively (Table 25).

Table 7. Number of herds according to their serologic status from 1994 to 1996.

Status	No.herds 12/94	No.herds 06/95	No.herds 12/95	No.herds 06/96	No.herds 10/96	Difference from 12/94 to 10/96
OADF	45	217	526	619	719	**674**
ADF	9	25	71	108	129	**120**
cOADF	290	434	268	213	169	**-121**
PS/cOADF	489	246	79	64	106	**-383**
cADF	47	101	67	55	40	**-7**
PS/cADF	218	111	36	18	28	**-210**
PS	1144	1319	1224	1193	1536	**392**
Non-operational	491	503	829	987	982	**491**
Total	2733	2956	3100	3257	3707	

cOADF= candidateOADF
cADF= candidate ADF

Table 7a. Linear regressions on months for total number of herds, and the categories OADF, ADF and PS herds.

Regression	N	Equation	F	P	Interpretation
Total herds	5	Y=2697.5 + 39.1X	29.1	<0.05	*
OADF	5	Y=60.4 + 31.4X	91.5	<0.01	**
ADF	5	Y=1.5 + 5.8X	167.8	<0.001	***
PS	5	Y=1156 + 10.9X	1.9	>0.05	NS

* highly significant
** very significant
*** extremely significant
NS non significant

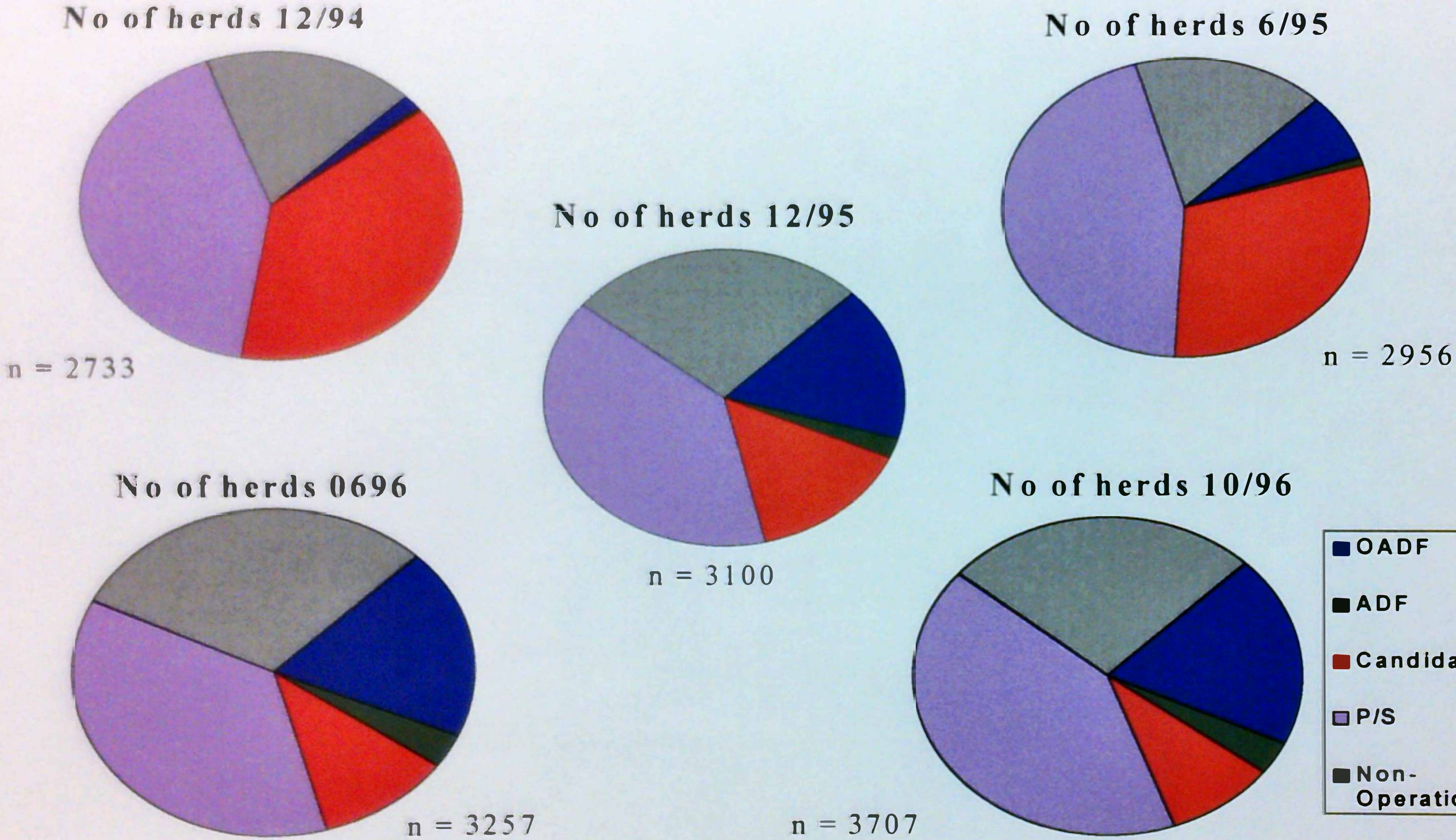


Figure 1. Number of herds according to their current status from 12/94 to 10/96.

Table 8. Number and proportion of PS herds by region serologically tested and infected by the end of 1996.

Region	% of PS herds	No.of herds blood surveyed during 1996	% Herds infected from 1996 survey *1993 in brackets*	Total number of PS herds within Division	Estimated number of **infected** herds
UK0	32%	14	21% [28%]	103	22
UK1	70%	49	22% [31%]	222	48
UK2	58%	21	9.5% [18%]	109	10
UK3	70%	71	24% [28%]	325	78
UK4	43%	72	29% [33%]	207	60
UK5	45%	7	57% [22%]	45	26
UK6	46%	22	4.5% [15%]	120	5
UK7	57%	20	30% [44%]	52	16
UK8	60%	30	3.3% [19%]	261	9
UK9	37%	20	15% [11%]	92	14
Total		326		1536	288

Percentage of infection 1993 vs 1996 by DVO

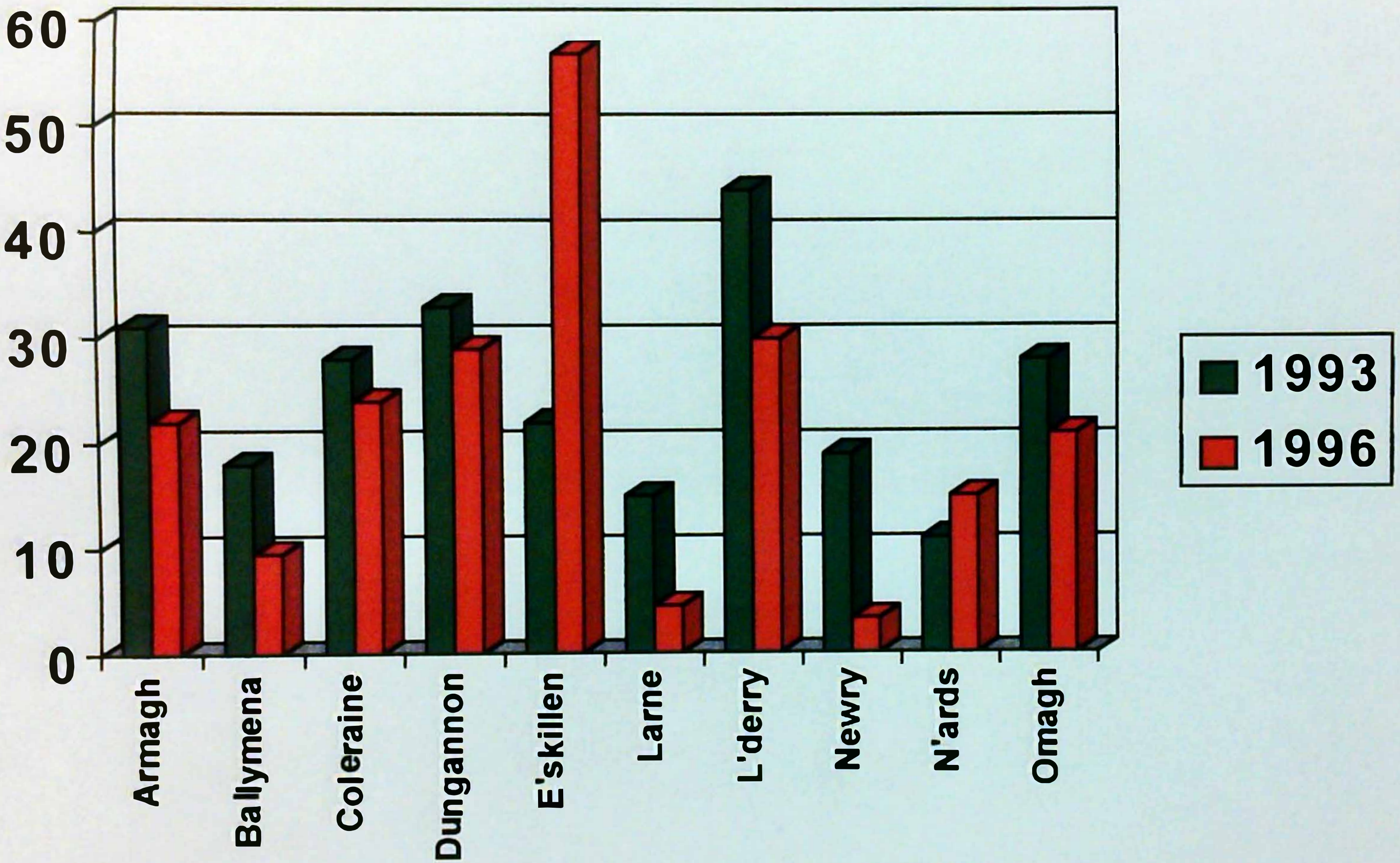


Fig 2 Percentage of infected herds from 1993 to 1996 by region

Table 9. Proportion of herds according to their serologic status from 1994 to 1996.

Health status	% herds 12/94	% herds 12/95	% herds 10/96
OADF	2%	23%	26%
ADF	0.4%	3%	4.7%
Candidate	46%	29%	13.3%
PS	51%	54%	56%
Total number of operational herds	2242	2271	2725

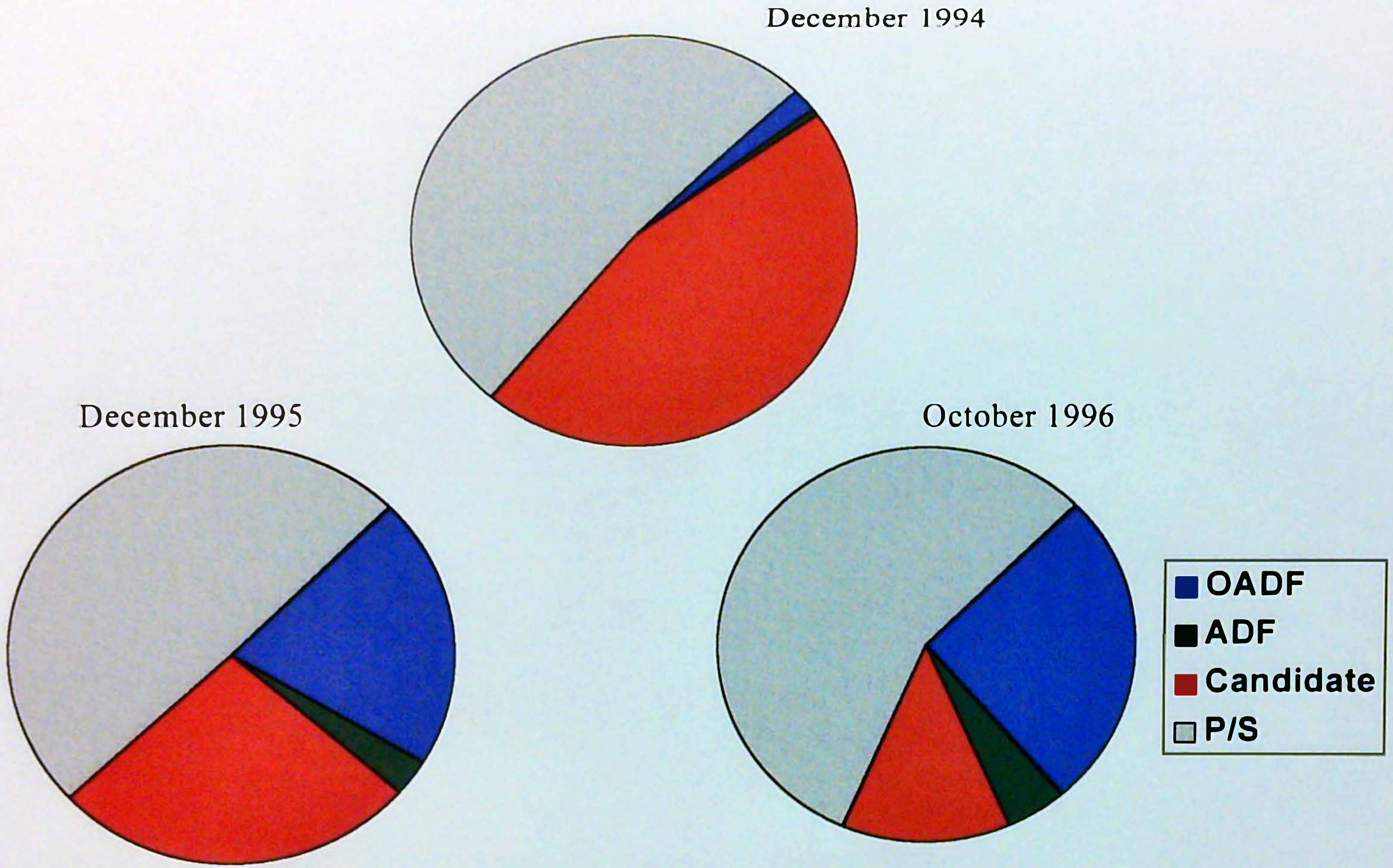
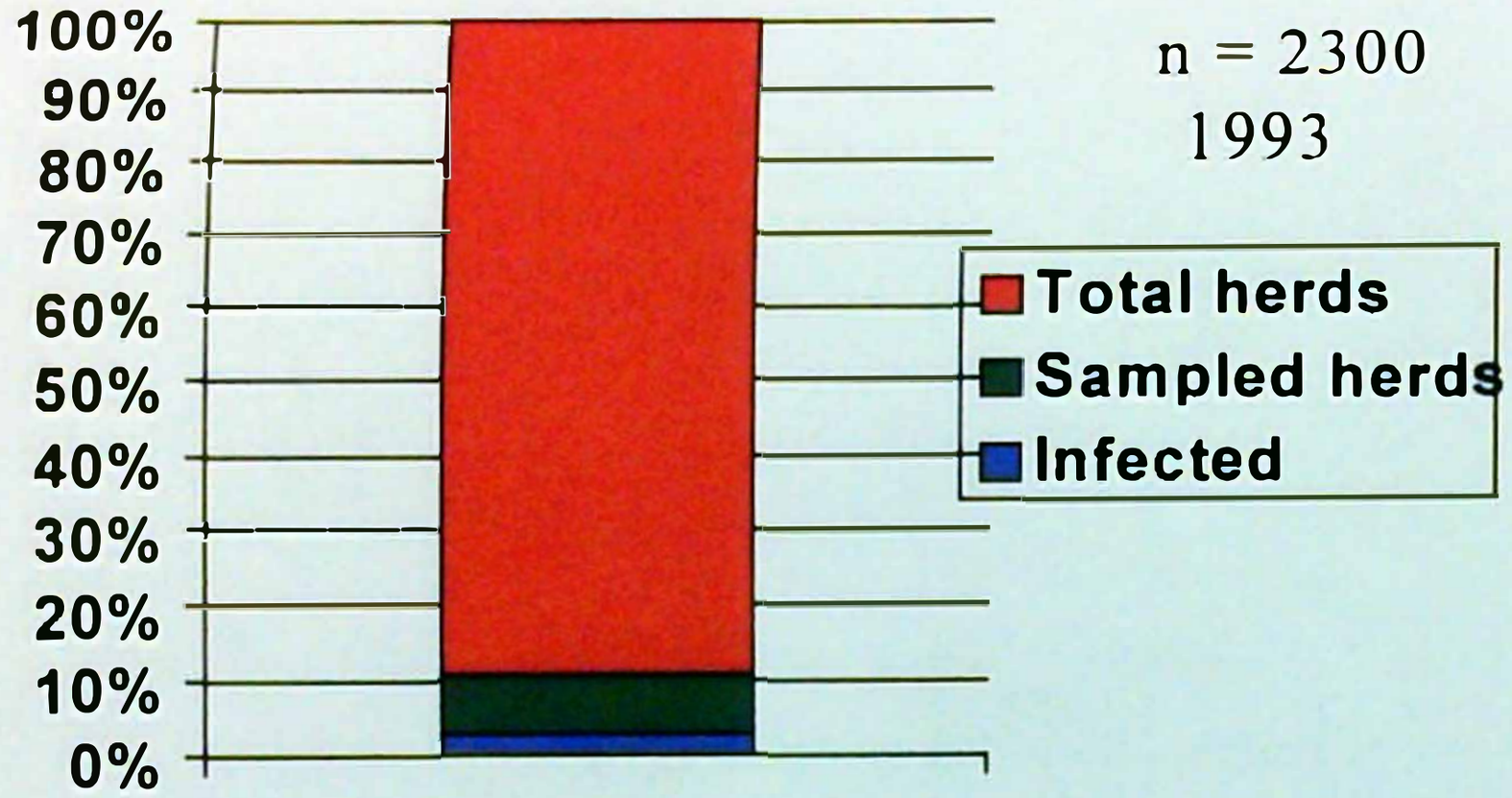
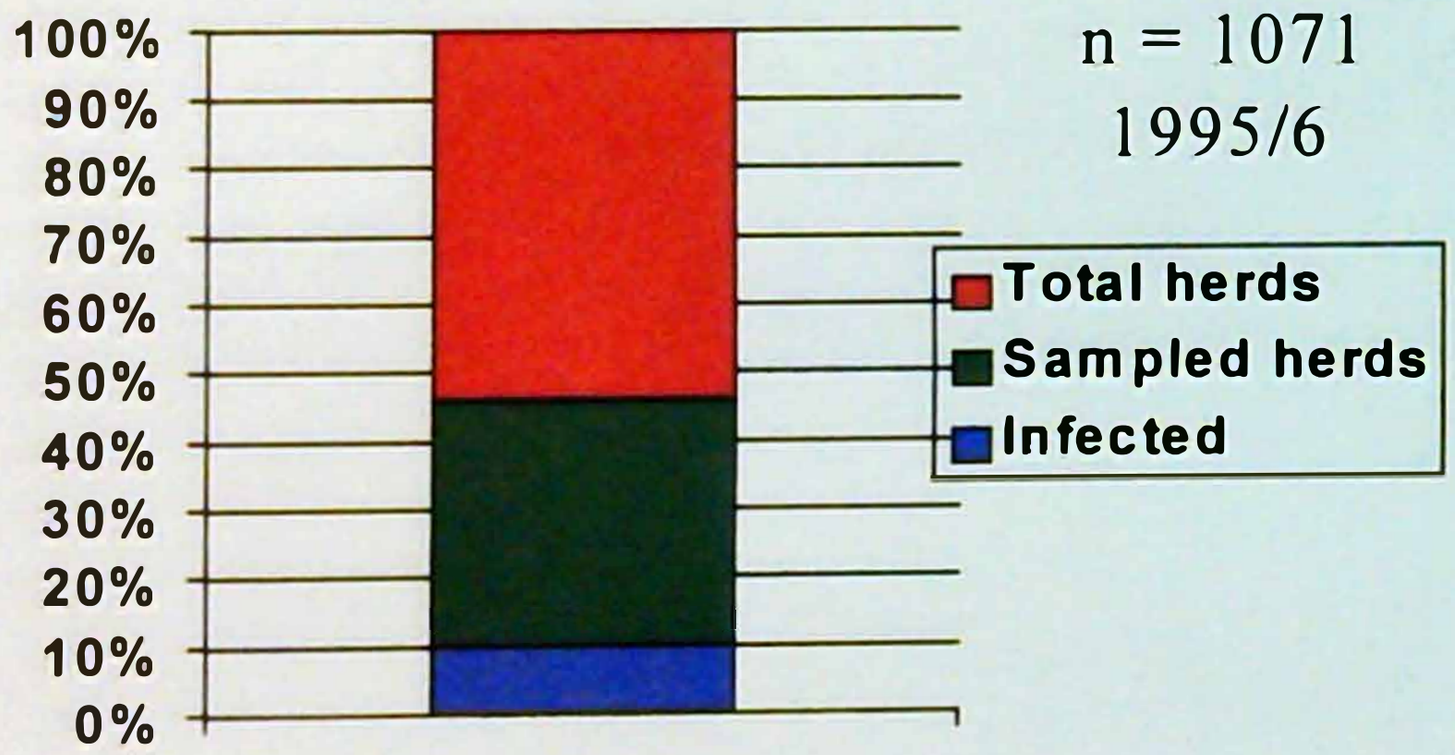


Figure 3. Proportion of herds according to the three main health status categories.

Table 10. Number and percentage of the condition of PS subcategory herds.

	PS herds= 1536 PS Subcategories				Total
	PS1	PS2	PS3	PS4	
Vaccination	+	+	-	-	
Infection	-	+	-	+	
Percentage of herds	34.4%	13%	39.6%	13%	
Number of sampled herds	178	67	205	67	517 (33.6%)





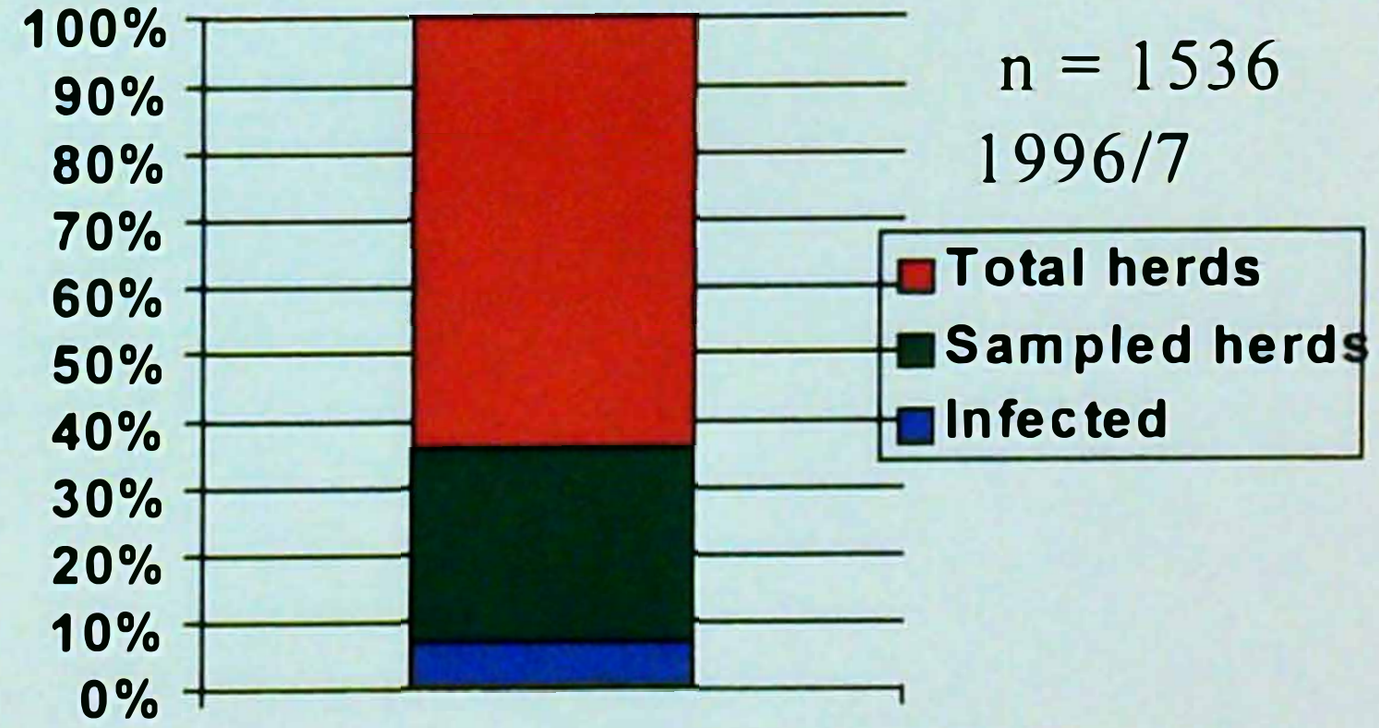


Figure 4 Proportion of antibody tesed and infected PS herds

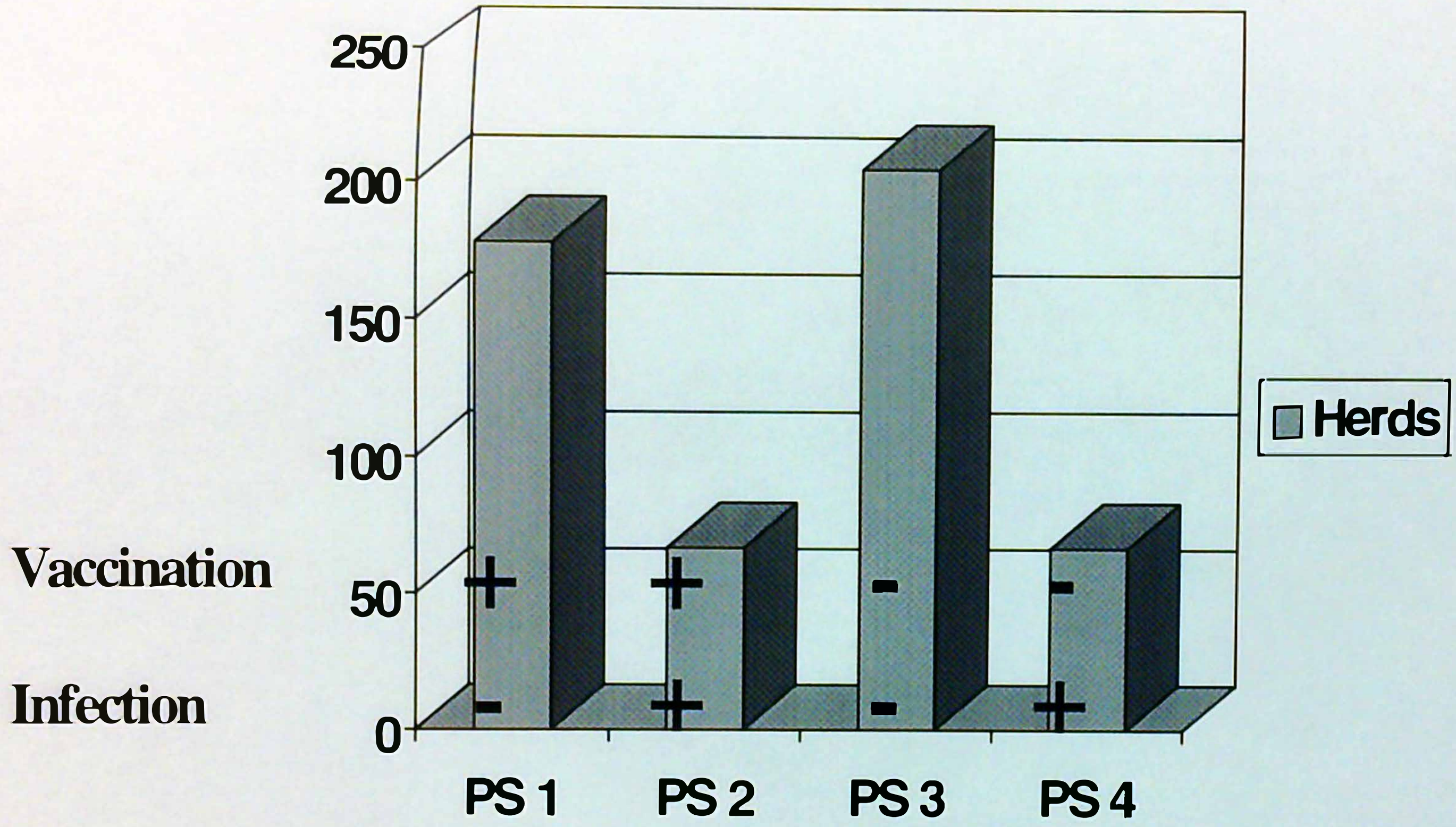


Figure 4a. Number of PS herds that were sampled by the end of 1996.

Table 11. Number and proportion of herds of **group A** by region.

Region	Number of herds	Poportion within the region
UKO	89	13.3 %
UK1	154	23.1 %
UK2	9	1.3 %
UK3	105	15.7 %
UK4	5	0.7 %
UK5	1	0.1 %
UK6	84	12.6 %
UK7	8	1.2 %
UK8	200	30.0 %
UK9	12	1.8 %
Total	**667**	**100 %**

Table 12. Number and proportion of herds of **group B** by region.

Region	Number of herds	Proportion within the region
UK0	21	2.5 %
UK1	6	0.7 %
UK2	90	10.7 %
UK3	176	21.0 %
UK4	211	25.1 %
UK5	52	6.2 %
UK6	28	3.3 %
UK7	52	6.2 %
UK8	99	11.8 %
UK9	105	12.5 %
Total	**840**	**100** %

Contour map of N Ireland showing Divisional Veterinary Areas

REGIONS

1. UK1 (Armagh)
2. UK2 (Ballymena)
3. UK3 (Coleraine)
4. UK4 (Dungannon)
5. UK5 (E'skillen)
6. UK6 (Larne)
7. UK7 (L'derry)
8. UK8 (Newry)
9. UK9 (N'ards)
10. UK0 (Omagh)

Map 1. Geographic distribution of herds by regions.

Key:

✝ = 10 herds (group A)

● = 10 herds (group B)

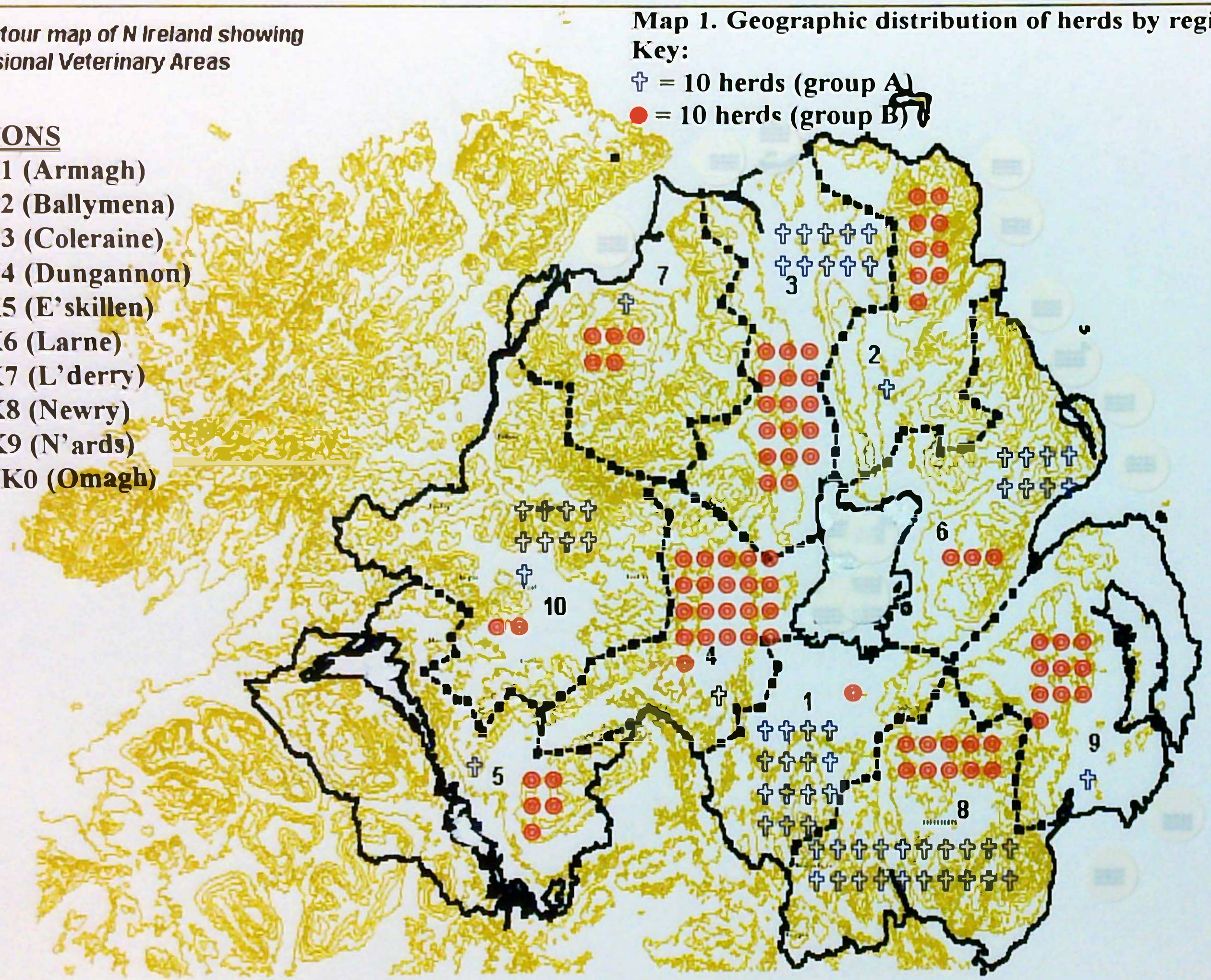


Table 13. Number and proportion of herds of **group A** by current health status.

Current health status	Number of herds	Proportion
ADF	42	6.3 %
CA	12	1.8 %
CO	44	6.6 %
OADF	133	19.9 %
PCA	1	0.1 %
PCO	4	0.6 %
PS	33	4.9 %
PS*	42	6.3 %
PS1	97	14.5 %
PS2	32	4.8 %
PS3	187	28.0 %
PS4	40	6.0 %
Total	**667**	**100** %

Table 14. Number of herds of **group B** by current health status.

Current health status	Number of herds	Proportion
ADF	0	0 %
CA	13	1.5 %
CO	43	5.1 %
OADF	0	0 %
PCA	2	0.2 %
PCO	42	5.0 %
PS	359	42.7 %
PS*	15	1.8 %
PS1	109	13.0 %
PS2	51	6.1 %
PS3	154	18.3 %
PS4	52	6.2 %
Total	**840**	**100** %

Table 15. Number of herds of **group A** by region and current health status.

	Current health status												
Region	**ADF**	**CA**	**CO**	**OADF**	**PCA**	**PCO**	**PS**	**PS***	**PS1**	**PS2**	**PS3**	**PS4**	**Total**
UK0	7	7	13	29	0	0	4	0	4	8	12	5	89
UK1	13	1	12	29	0	3	14	37	14	6	20	5	**154**
UK2	0	0	0	1	0	0	0	0	3	1	4	0	9
UK3	7	2	3	26	0	0	1	1	14	7	37	7	**105**
UK4	1	1	0	1	0	0	1	0	1	0	0	0	5
UK5	0	0	1	0	0	0	0	0	0	0	0	0	1
UK6	9	0	0	15	0	0	4	2	11	2	33	8	84
UK7	0	0	1	1	0	0	0	0	0	1	4	1	8
UK8	5	1	10	27	1	1	9	2	49	7	75	13	**200**
UK9	0	0	4	4	0	0	0	0	1	0	2	1	12
Total	**24**	**12**	**44**	**133**	**1**	**4**	**33**	**42**	**97**	**32**	**187**	**40**	667

Table 15a. Number and proportion of herds of the groups ***Others*** and ***PS's*** of **group A** by region and current health status.

	Current health status								
Region	***Others***[1] (%)	PS (%)	PS* (%)	PS1 (%)	PS2 (%)	PS3 (%)	PS4 (%)	**Total *PS's***[2] (%)	**TOTAL**
UK0	*56 (62.9)*	4 (4.4)	0	4 (4.4)	8 (8.8)	12 (13.4)	5 (5.6)	*33 (37.0)*	89
UK1	*58 (37.6)*	14 (9.0)	37 (24.0)	14 (9.0)	6 (11.1)	20 (12.9)	5 (3.2)	*96 (62.3)*	**154**
UK2	*1 (11.1)*	0	0	3 (33.3)	1 (11.1)	4 (44.4)	0	*8 (88.8)*	9
UK3	*38 (36.1)*	1 (0.9)	1 (0.9)	14 (13.3)	7 (6.6)	37 (35.2)	7 (6.6)	*67 (63.8)*	**105**
UK4	*3 (60.0)*	1 (20)	0	1 (20)	0	0	0	*2 (40.0)*	5
UK5	*1 (100)*	0	0	0	0	0	0	*0*	1
UK6	*22 (26.1)*	4 (4.7)	2 (2.3)	11 (13.0)	2 (2.3)	33 (39.2)	8 (9.5)	*62 (73.8)*	84
UK7	*2 (25)*	0	0	0	1 (12.5)	4 (50)	1 (12.5)	*6 (75.0)*	8
UK8	*45 (22.5)*	9 (4.5)	2 (1.0)	49 (24.5)	7 (3.5)	75 (37.5)	13 (6.5)	*155 (77.5)*	**200**
UK9	*8 (66.6)*	0	0	1 (8.3)	0	2 (16.6)	1 (8.3)	*4 (33.3)*	12
Total	*236 (35.3)*	33 (4.9)	42 (6.2)	97 (14.5)	32 (4.7)	187 (28.0)	40 (5.9)	*431 (64.6)*	667

[1] *Others* include: ADF, CA, CO, OADF, PCA and PCO herds.
[2] *PS's* include: PS, PS*, PS1, PS2, PS3 and PS4 herds.

Table 15b. P values of the comparison in the numbers of herds by region and current health status between the groups ***Others*** and ***PS's***, and *PS's* (one to each other) of **group A** by chi square test.

	Herd type comparison						
Values	***Others*** and ***PS's***	PS1 and PS2	PS3 and PS4	PS1 and PS3	PS2 and PS4	PS1+PS2 and PS3+PS4	PS1+PS3 and PS2+PS4
chi square	66.01	15.2	2.46	6.15	4.87	5.98	14.69
df	5	4	4	4	3	5	4
P	0.001 ***	0.001 ***	0.65 NS	0.18 NS	0.18 NS	0.38 NS	0.001 ***

*** extremely significant
NS non significant

Contour map of N Ireland showing Divisional Veterinary Areas

REGIONS

1. UK1 (Armagh)
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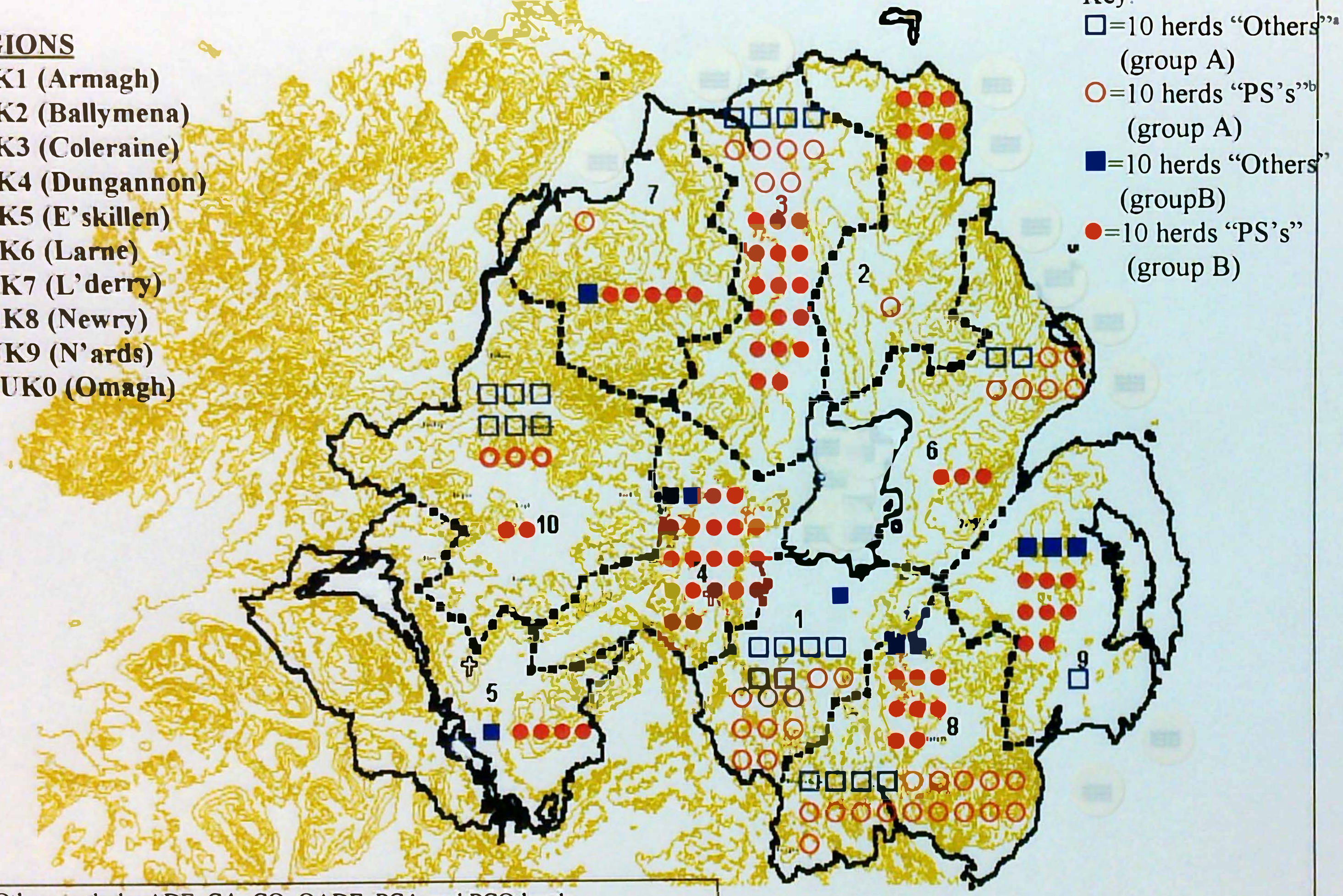
5. UK5 (E'skillen)
6. UK6 (Larne)
7. UK7 (L'derry)
8. UK8 (Newry)
9. UK9 (N'ards)
10. UK0 (Omagh)

Map 2. Geographic distribution of herds from group A and B by region and current status.



a. Others include: ADF, CA, CO, OADF, PCA and PCO herds.
b. PS's include: PS, PS*, PSI, PS2, PS3 and PS4 herds

Table 16. Number of herds of **group B** by region and current health status.

	Current health status												
Region	**ADF**	**CA**	**CO**	**OADF**	**PCA**	**PCO**	**PS**	**PS***	**PS1**	**PS2**	**PS3**	**PS4**	**Total**
UK0	0	1	3	0	0	0	17	0	0	0	0	0	21
UK1	0	0	0	0	0	6	0	0	0	0	0	0	6
UK2	0	0	0	0	0	1	30	2	26	4	21	6	90
UK3	0	1	2	0	0	2	34	2	32	17	63	23	**176**
UK4	0	6	9	0	0	7	100	1	32	26	22	8	**211**
UK5	0	1	3	0	0	10	22	1	3	0	11	1	52
UK6	0	0	0	0	0	1	23	0	2	0	2	0	28
UK7	0	1	1	0	2	1	22	1	1	2	14	7	52
UK8	0	0	14	0	0	5	73	4	1	0	2	0	99
UK9	0	3	11	0	0	15	32	4	12	2	19	7	**105**
Total	**0**	**13**	**43**	**0**	**2**	**42**	**359**	**15**	**109**	**51**	**154**	**52**	840

Table 16a. Number and proportion of herds of the groups ***Others*** and ***PS's*** of **group B** by region and current health status.

	Current health status								
Region	***Others***[1] (%)	**PS** (%)	**PS*** (%)	**PS1** (%)	**PS2** (%)	**PS3** (%)	**PS4** (%)	**Total *PS's*[2]** (%)	**TOTAL**
UK0	*4 (19.0)*	17 (80.0)	0	0	0	0	0	*17 (80.0)*	21
UK1	*6 (100.0)*	0	0	0	0	0	0	*0*	6
UK2	*1 (1.1)*	30 (33.3)	2 (2.2)	26 (28.8)	4 (4.4)	21 (23.3)	6 (6.6)	*89 (98.8)*	**90**
UK3	*2 (2.8)*	34 (19.3)	2 (1.1)	32 (16.5)	17 (9.6)	63 (35.7)	23 (13.0)	*171 (97.1)*	**176**
UK4	*22 (10.4)*	100 (47.3)	1 (0.4)	32 (15.1)	26 (12.3)	22 (10.4)	8 (3.7)	*189 (89.5)*	211
UK5	*14 (26.9)*	22 (42.3)	1 (1.9)	3 (5.7)	0	11 (21.1)	1 (1.9)	*38 (73.0)*	52
UK6	*1 (3.5)*	23 (82.1)	0	2 (7.1)	0	2 (7.1)	0	*27 (96.4)*	28
UK7	*5 (9.6)*	22 (42.3)	1 (1.9)	1 (1.9)	2 (3.8)	14 (26.9)	7 (13.4)	*47 (90.3)*	52
UK8	*19 (19.1)*	73 (73.7)	4 (4.0)	1 (1.0)	0	2 (2.0)	0	*80 (80.8)*	99
UK9	*29 (27.6)*	32 (30.4)	4 (3.8)	12 (11.4)	2 (1.9)	19 (18.0)	7 (6.6)	*76 (72.3)*	**105**
Total	*100 (11.9)*	359 (42.7)	15 (1.7)	109 (12.9)	51 (6.0)	154 (18.3)	52 (6.1)	*740 (88.0)*	840

[1] *Others* include: ADF, CA, CO, OADF, PCA and PCO herds.
[2] *PS's* include: PS, PS*, PS1, PS2, PS3 and PS4 herds.

Table 16b. P values of the comparison in the number of herds by region and current health status between the groups ***Others*** and ***PS's***, and *PS's* (one to each other) herds of **group B** by chi square test.

	Herd type comparison						
Values	*Others* and *PS's*	PS1 and PS2	PS3 and PS4	PS1 and PS3	PS2 and PS4	PS1+PS2 and PS3+PS4	PS1+PS3 and PS2+PS4
chi square	51.91	6.50	0.34	17.88	6.74	37.04	6.48
df	5	2	4	4	2	4	4
P	0.001 ***	0.03 *	0.98 NS	0.001 ***	0.03 *	0.001 ***	0.16 NS

* highly significant *** extremely significant

NS non significant

Table 16c. P values of the comparison in the number of herds by current health status between the groups [***Others*** and ***PS's***], [PS1+PS2 and PS3+PS4], and [PS1+PS3 and PS2+PS4] of **group A** and **Group B** together by chi square test.

	Herd type comparison		
Groups A + B	*Others* and *PS's*	PS1+PS2 and PS3+PS4	PS1+PS3 and PS2+PS4
Chi-square	116.93	3.73	5.68
df	1	1	1
P	0.001 ***	0.05 *	0.01 **

* highly significant
** very significant.
*** extremely significant.

Table 17. Comparison in the number of herds by current health status between the groups [***Others*** and ***PS's***] inter-regionally (one to each other), and intra-regionally of **group A** and **group B** by chi square test.

	Region group B									
Region group A	**UK0**	**UK1**	**UK2**	**UK3**	**UK4**	**UK5**	**UK6**	**UK7**	**UK8**	**UK9**
UK0	***(1) χ^2=11.4 P<0.001	***	***	***	***	***	NS	***	***	***
UK1		**(1) χ^2=6.93 P<0.003	***	***	***	***	***	***	***	***
UK2			NS(1)	***	**	NS	***	NS	***	NS
UK3				***(1) χ^2=62.2 P<0.001	***	***	***	***	***	***
UK4					**(1) χ^2=7.3 P<0.01	NS	***	***	***	***
UK5						NS(1)	***	**	***	**
UK6							*(1) χ^2=5.2 P<0.02	***	NS	***
UK7								NS(1)	***	NS
UK8									NS(1)	***
UK9										**(1) χ^2=5.9 P<0.009

* highly significant
** very significant
*** extremely significant
NS non significance
(1) difference between [***Others*** and ***PS's***] intra-regionally

Table 18. Number and proprotion of herds of **group A** by herd type.

Herd type	Number of herds	Proportion
A	4	1.1 %
BB	78	22.4 %
BBW	5	1.4 %
BW	227	65.2 %
F	34	9.8 %
Total	**348**	**100 %**

Herd type:A= Alterer or Improver, BB= Birth to Bacon, BBW= Birth to Bacon sells weaners, BW= Birth to Weaning, F= Fattener.

Table 19. Number of herds of **group A** by current health status and herd type.

	Herd type					
Current status	**A**	**BB**	**BBW**	**BW**	**F**	**Total**
ADF	0	0	0	1	0	**1**
CA	0	1	0	1	0	**2**
CO	0	8	0	17	3	**28**
OADF	0	0	0	1	0	**1**
PCA	0	0	0	0	0	**0**
PCO	0	0	1	2	1	**4**
PS	0	9	0	11	1	**21**
PS*	0	13	0	22	1	**36**
PS1	1	12	1	60	3	**77**
PS2	0	4	0	15	1	**20**
PS3	3	27	3	77	22	**132**
PS4	0	4	0	20	2	**26**
TOTAL	**4**	**78**	**5**	**227**	**34**	**348**

Herd type:A= Alterer or Improver, BB= Birth to Bacon, BBW= Birth to Bacon sells weaners, BW= Birth to Weaning, F= Fattener.

Table 19a. Number and proportion of herds of the groups ***Others*** and ***PS's*** of **group A** by current health status and herd type.

	Herd type					
Current status	**A (%)**	**BB (%)**	**BBW (%)**	**BW (%)**	**F (%)**	**Total (%)**
Others[1]	*0*	*9 (11.5)*	*1 (20)*	*22 (9.6)*	*4 (11.7)*	*36 (10.3)*
PS		9 (11.5)	0	11 (4.8)	1 (2.9)	21 (6.0)
PS*	0	13 (16.6)	0	22 (9.6)	1 (2.9)	36 (10.3)
PS1	1 (25)	12 (15.3)	1 (20)	60 (26.4)	3 (8.8)	77 (22.1)
PS2	0	4 (5.1)	0	15 (6.6)	1 (2.9)	20 (5.7)
PS3	3 (75.0)	27 (34.6)	3 (60)	77 (33.7)	22 (64.7)	132 (37.9)
PS4	0	4 (5.1)	0	20 (8.8)	2 (5.8)	26 (7.4)
Total ***PS's***[2] **(%)**	*4 (100.0)*	*69 (88.4)*	*4 (80)*	*205 (90.3)*	*30 (88.2)*	*312 (89.6)*
TOTAL	**4**	**78**	**5**	**227**	**34**	**348**

[1] *Others* include: ADF, CA, CO, OADF, PCA and PCO herds.

[2] *PS's* include: PS, PS*, PS1, PS2, PS3 and PS4 herds.

Herd type: A= Alterer or Improver, BB= Birth to Bacon, BBW= Birth to Bacon sells weaners, BW= Birth to Weaning, F= Fattener.

Table 20. Number and proportion of herds of **group B** by herd type.

Herd type	Number of herds	Proportion
A	1	0.4 %
BB	86	31.9 %
BBF	1	0.4 %
BBW	3	1.1 %
BW	147	54.4 %
F	30	11.1 %
PD	1	0.4 %
SD	1	0.4 %
Total	**270**	**100 %**

Herd type: A= Alterer or Improver, BB= Birth to Bacon, BBW= Birth to Bacon sells weaners, BW= Birth to Weaning, F= Fattener, PD= Dealer Production Pigs, SD= Dealer (Sows).

Table 21. Number of herds of **group B** by current health status and herd type.

	Herd type								
Current health status	**A**	**BB**	**BBF**	**BBW**	**BW**	**F**	**PD**	**SD**	**TOTAL**
ADF	0	0	0	0	0	0	0	0	**0**
CA	0	2	0	0	1	0	0	0	**3**
CO	0	3	0	0	1	1	0	0	**5**
OADF	0	0	0	0	0	0	0	0	**0**
PCA	0	0	0	0	1	0	0	0	**1**
PCO	0	3	0	0	3	0	0	0	**6**
PS	0	2	0	0	8	0	0	0	**10**
PS*	0	2	0	0	2	0	0	0	**4**
PS1	0	19	0	0	40	9	1	0	**69**
PS2	0	11	0	0	13	2	0	0	**26**
PS3	1	36	1	2	56	10	0	0	**106**
PS4	0	8	0	1	22	8	0	1	**40**
TOTAL	**1**	**86**	**1**	**3**	**147**	**30**	**1**	**1**	**270**

Herd type: A= Alterer or Improver, BB= Birth to Bacon, BBW= Birth to Bacon sells weaners, BW= Birth to Weaning, F= Fattener, PD= Dealer Production Pigs, SD= Dealer (Sows).

Table 21a. Number and proportion of herds of groups ***Others*** and ***PS's*** of **group B** by current health status and herd type.

	Herd type								
Current health status	**A (%)**	**BB (%)**	**BBF (%)**	**BBW (%)**	**BW (%)**	**F (%)**	**PD (%)**	**SD (%)**	**TOTAL**
Others[1]	*0*	*8 (9.3)*	*0*	*0*	*6 (4.0)*	*1 (3.3)*	*0*	*0*	*15 (5.5)*
PS	0	2 (2.3)	0	0	8 (12.2)	0	0	0	10 (3.9)
PS*	0	2 (2.3)	0	0	2 (1.3)	0	0	0	4 (1.5)
PS1	0	19 (22.0)	0	0	40 (27.2)	0 (30.0)	1 (100.0)	0	69 (25.5)
PS2	0	11 (12.7)	0	0	13 (8.8)	2 (6.6)	0	0	26 (9.6)
PS3	1 (100.0)	36 (41.8)	1 (100.0)	2 (66.6)	56 (38.0)	10 (33.3)	0	0	106 (39.2)
PS4	0	8 (9.3)	0	1 (33.3)	22 (14.9)	8 (26.6)	0	1 (100.0)	40 (14.8)
Total *PS's*[2] (%)	*1 (100.0)*	*78 (90.6)*	*1 (100.0)*	*3 (100.0)*	*141(95.9)*	*29 (96.6)*	*1 (100.0)*	*1 (100.0)*	*255 (94.4)*
Total	**1**	**86**	**1**	**3**	**147**	**30**	**1**	**1**	**270**

[1]Others include: ADF, CA, CO, OADF, PCA and PCO herds.

[2]PS's include: PS, PS*, PS1, PS2, PS3 and PS4 herds.[1]Others include: ADF, CA, CO,

Herd type:A= Alterer or Improver, BB= Birth to Bacon, BBW= Birth to Bacon sells weaners, BW= Birth to Weaning, F= Fattener, PD= Dealer Production Pigs, SD= Dealer (Sows).

Table 22. Pig population of **group A** by region.

	Region									
Herdsize	UK0	UK1	UK2	UK3	UK4	UK5	UK6	UK7	UK8	UK9
Mean	385.6	255.9	185.6	276.1	106		356.1	19.2	172.5	24.1
Median	36	74	97	63	78	1	80.5	15	30	10.5
Range	1-3282	1-1756	9-491	1-1938	58-422	1	1-516	5-41	1-2118	2-73
Sum	22749	37611	1299	20709	558	1	28135	154	31744	193
N	59	147	7	75	3	1	79	8	184	8

Table 23. Pig population of **group B** by region.

	Region									
	UK0	UK1	UK2	UK3	UK4	UK5	UK6	UK7	UK8	UK9
Mean	93.6	13.7	300.4	334.6	378.6	127.9	40.2	131.1	136.8	193.7
Median	56.5	12	52	70	81	12	24	25	31	23.5
Range	1-1-741	2-27	1-2804	2-4056	1-9999	1-1800	1-300	1-2155	1-2647	1-3349
Sum	690	41	22828	27102	52628	6395	803	6425	10674	17918
N	7	3	76	81	131	50	20	49	78	92

Table 23a. Intra-regional comparison of the pig population of **group A** and **group B** by Mann-Whitney test.

	Comparison									
Values	UK0 and UK0	UK1 and UK1	UK2 and UK2	UK3 and UK3	UK4 and UK4	UK5 and UK5	UK6 and UK6	UK7 and UK7	UK8 and UK8	UK9 and UK9
Mann-Whitney	230.5	68.5	246	3017	1830	-	413	156.5	6938	258.5
P	0.92	0.04	0.74	0.94	0.72	-	0.001	0.37	0.79	0.16
	NS	*	NS	NS	NS	-	***	NS	NS	NS

* highly significant

*** extremely significant

NS non significance

Table 24. Pig population of **group A** by current health status.

	Current health status													
	ADF	CA	CO	OADF	PCA	PCO	PS	PS*	PS1	PS2	PS3	PS4	Others	PS's
Mean	512.4	464.5	117.2	164.7	459.5	184.9	324.1	264.2	208.6	469.1	237.4	337.9		
Median	212	157.5	35	52	200.5	65	86.5	79	21	105	44.5	55.5		
Range	5-3282	1-3282	2-1628	1-2156	5-1756	1-2156	1-1963	31756	1-2181	3-2000	1-4500	1-5116		
Sum	19984	8361	8670	19434	20219	13685	9725	10040	18145	12666	37509	11488	90353	99573
N	39	18	74	118	44	74	30	38	87	27	158	34	367	374

Table 25. Pig population of **group B** by current health status.

	Current health status													
	ADF	CA	CO	OADF	PCA	PCO	PS	PS*	PS1	PS2	PS3	PS4	Others	PS's
Mean		238.5	130.6		21	58.6	139.7	339.5	248.4	419.8	369.2	570.9		
Median		52	33		21	16	29	129	52	88.5	55	73.5		
Range		10-1174	1-1223		13-29	1-708	0-3549	16-1970	1-4742	3-2804	1-4056	7-9999		
Sum		2624	4050		42	1757	32551	4413	22105	15954	41357	20551	8473	136951
N		11	31		2	30	233	13	89	38	112	36	74	521

5. DISCUSSION

The purpose of this study was to investigate the progress of the AD scheme running officially in Northern Ireland from September 1994, and to analyse the difference between herds by region, current health status, herd type and pig population.

The progress of the AD scheme in Northern Ireland was measured by increase in the number of herds through the time. More herds having high health status have been increased progressively every six months (Tables 7 and 7a). The significant difference of the linear regression for the high health status (OADF, ADF) support this. However, the PS herds have increased their number as well which means that more new herds have joined the scheme; unfortunately no sampling enough has been practised in such herds because the "BSE crisis". In other words, more herds had higher health status in 1996 than before in 1994 which means that the worry of getting AD disease in pig operations has decreased; otherwise, farmers would be reluctant to get into the pig industry, but it might be coupled to the fact of the "BSE crisis" which created a change into pig farming rather than cattle farming.

The main significant differences in the number of herds were the comparisons between the groups 1) ***Others*** and ***PS's***, 2)PS1+PS2 and PS3+PS4, and 3)PS1+PS3 and PS2+PS4 (Table 16c). In other words, there were significant differences in the number of herds between 1) free herds and non-free herds, 2) vaccinated herds and non-vaccinated herds, and 3) infected herds and non-infected herds in **group A** and **group B** when compared together. The results were expected and are similar to those reported differences between vaccinated and non-vaccinated herds (Thawley and Morrison, 1989; Stegeman et al, 1996; Houben, Dijkhuizen, De Jong and others,

1993).

In contrast, significant comparisons were only detected between ***Others*** and ***PS's*** when **group A** and **group B** were compared separately. The other comparisons were not consistent since the comparison between PS's (one to each other) resulted in a cross-over difference. Unexpectedly, the comparison between PS1+PS3 and PS2+PS4 was significantly different for **group A** whereas the same comparison was not significant in **group B**. Consequently the interpretation is confused and requires more attention.

UK3 and UK4 which share borders were the most important regions in terms current health status and pig population since half of PS2 and PS4 (infected) herds were found in these two regions, specially in region UK3. The infected herds are geographically clustered creating high pig density areas that facilitates the spread of the disease (Weigel, Austin, Seigel and *others*, 1992; Norman *et al*, 1996; Austin *et al*, 1992; Auvigne and Hery, 1997). Airborne transmission of AD has to be taken into account in eradication programmes, specially in areas with high density and high prevalence of the disease (Scheidt, Reuff, Grant and *others*, 1991). This can be applied to the fact that regions UK3 and UK4 share borders and belong to the main pig production area of Northern Ireland where aerosol transmission might be possible between them.

BW being the most common herd type has the advantage of rearing fewer pigs on the same site, reducing the chances of virus circulation in fattening pigs because pigs are sold at weaning. This limitates the size and composition of the susceptible population (Leontides, Ewald, Mortensen and *others*, 1995). Further

studies should be done to identify if seropositive pigs belong to either the breeding stock, or fattening pigs, or both. On the other hand, BW has the disadvantage of increasing the chances of infection by pig movements when weaners are sold, since 22 % of these herds were regarded as infected. Other control measures together with vaccination should be practised to prevent reintroduction of virus from outside, for instance pig movement control (Stegeman, 1997).

Infected herds had a mean pig population not greater than 571 pigs and were located basically in two regions. However, Bouma, De Jong and Kimman (1995) believed that the transmission of AD did not depend on herd size and they postulated that the virus might even spread in small populations.

An association between the spread of AD among breeding pigs and the presence of non-vaccinated pigs has been reported (Duffy et al, 1991; Morrison, Marsh, Anderson and *others*, 1991). The number of infected, unvaccinated herds reported in this study was 92 of which 41 % were found in regions UK3 and UK4. This could be a point to take into account to encourage the vaccination of these herds.

More attention should be focused on PS herds since no antibody testing has been done so far. This is important to determine their proper current health status since there is a considerable number (392) of herds which represents 10.7% of the total number of herds of which 37% were found in regions UK3 and UK4. PS herds must be regarded as the high risk group.

Further studies should be done based on economical aspects, taking into account the prevalence of the disease, the distribution of infected herds, and the willingness of producers who own free AD pig farms to pay compensation of infected herds. Having found that infected herds are located mainly in a certain area, it would be helpful to determine what is more economically feasible whether to apply a stamping out policy or to keep on with the vaccination strategy.

The procedure of antibody testing of the AD scheme in Northern Ireland is based on testing the positive samples to the whole ELISA with one differential ELISA (gE). If these positive samples give a positive result to the ELISA (gE) then they are tested again. In other countries such as the Netherlands seropositive sera to the ELISA (gE) are tested again twice using two different commercial ELISA (gE) tests to verify the positive reactors (Van Nes *et al*, 1997). This laboratory strategy is to be recommended in eradication programmes when the prevalence of the disease is low and the diagnosis requires to be more precise.

The AD scheme in Northern Ireland has the advantage of being financially supported by the State which provides the antibody-testing free of cost for producers whereas in other European countries such as Sweden pig producers have to bear the costs of getting an AD free status (Engel *et al*, 1989).

The database currently used in the eradication programme of the disease in Northern Ireland is efficient since it is regularly updated, and every pig herd in the country is recorded. This may account as another advantage of the eradication

programme. However, it has to be improved in terms of having a bigger set of records of herd characteristics, since for instance, only 51 % and 32 % of herds of **group A** and **group B** respectively had recorded the herd type. The same gap of records may also be applied to pig population. This might be useful for further epidemiological studies to identify the risk factors of AD in Northern Ireland.

The current eradication scheme in Northern Ireland is making progress since the prevalence of the disease has dropped from 25 % to 10.7 % infected herds after two years of the compulsory vaccination programme. Similar results of an area-wide (96 km^2) intensive vaccination programme in the Netherlands also reported a marked reduction of the prevalence after two years using similar vaccination schedule (Stegeman *et al*, 1997). However, failure in the eradication of the disease using a vaccination programme based on gene-deleted vaccine has been reported (Kavanagh, 1996).

6. CONCLUSIONS

AD is an important disease affecting the good performance of the pig production in many countries. Few countries have achieved eradication whereas many others are currently applying programmes to eradicate the disease. The scheme in Northern Ireland has made progress.

One of the major obstacles to clean AD from pig farms is the fact that ADV causes latent infections which are not clinically manifested from which the virus can be reactivated under certain conditions.

The development of gene-deleted vaccines coupled with the complementary serologic tests which differentiate vaccinated from infected pigs are very good tools for monitoring the progress achieved in eradication programmes. Vaccination against AD is the main control measure applied in several countries which are trying to eradicate the disease

Risk factors related to the presentation and perpetuation of ADV infection include environmental and herd characteristics features that interact together making more difficult the control of the disease.

The distribution of AD in Northern Ireland showed that the disease was more frequent in regions UK3 and UK4. More than 50 % of the infected herds were located in these two regions.

The AD eradication scheme in Northern Ireland had the advantages of dealing with a considerable low prevalence of the disease and pig population, regionally limited infection, positive attitude of pig producers, and official funding support. These facts, coupled with accurate diagnostic tests and marker vaccines that enable the differentiation of vaccinated from infected pigs, and the current knowledge on the

epidemiology of ADV make likely its eradication from this country.

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Appendix 1. Northern Ireland regions (DVO divisions)

Omagh	UK0
Armagh	UK1
Ballymena	UK2
Coleraine	UK3
Dungannon	UK4
Enninskillen	UK5
Larne	UK6
L'derry	UK7
Newry	UK8
N'ards	UK9

Appendix 2. Current health status classification.

ADF: Aujeszky's disease free.

CA: candidate ADF.

CO: candidate OADF.

OADF: officially Aujeszky's disease free.

PCA: not yet tested for antibodies, may have vaccinated pigs, candidate ADF.

PCO: not yet tested for antibodies, no vaccination, candidate OADF.

PS: default status when a herd is registered and no information of its health status is known.

PS*: downgraded herd but it may go up again.

PS1: all tested pigs show evidence of vacccination but no evidence of infection.

PS2: all tested pigs show evidence of vaccination and some evidence of infection.

PS3: not all tested pigs show evidence of vaccination but not apparent infection.

PS4: not evidence of vaccination or infection.

Appendix 3. Herd type classification.

A	Alterer or Improver
BB	Birth to Bacon
BBF	Birth to Bacon buys Weaners
BBW	Birth to Bacon sells weaners
BW	Birth to Weaning
F	Fattener
PD	Dealer Production Pigs
SD	Dealer (Sows)